**Focal symptoms in general paralysis / by C. Macfie Campbell.**

From the PSYCHIATRIC INSTITUTE of the
NEW YORK STATE HOSPITALS

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IN

# GENERAL PARALYSIS

—BY—

C. MACFIE CAMPBELL, M. D., B. Sc.,

Associate Professor of Psychiatry,
Johns Hopkins University, Baltimore

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# INDEX

# FOCAL SYMPTOMS IN GENERAL PARALYSIS.*

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## General Introduction.

The opening years of the twentieth century have proved to be an exceedingly fruitful period for the study of general paralysis of the insane. In the nineteenth century general paralysis was early recognized by the French school to be a more or less independent disease with a well-defined symptomatology and characteristic course (Esquirol, Bayle, Calmeil). The etiological importance of syphilis gradually won recognition on statistical grounds; the unsatisfactory nature of this form of proof, however, was sufficiently clear from the extraordinary divergence of the statistics furnished by the various authors. The pathological anatomy of the disease gave rise to much controversy from an early date. In the same year, 1826, Bayle and Calmeil, both drawing their material from Charenton, published works on general paralysis. For Bayle the essence of the disorder is a chronic meningitis, the disease is referred to as "insanity with incomplete paralysis due to chronic meningitis;" Calmeil, commenting with some severity on the views of Bayle, maintains that the morbid process is essentially a chronic encephalitis, an inflammation which especially involves the surface of the brain and the meninges. In the second half of the nineteenth century the presence of a degenerative and of an inflammatory element in the pathological picture was recognized, and the question was keenly debated which of these two elements was primary; an exact analysis of the inflammatory element is a comparatively recent achievement.

* Thesis presented in July, 1911, to the University of Edinburgh for the degree of M. D.

The very important progress which the pathological anatomy of general paralysis has made within recent years is well illustrated by the fact that, as late as 1896, Nissl was making his pathological diagnosis of general paralysis on the basis of his general impression of the anatomical picture, without clear insight into the differential histopathological details; what is more, his statements at that date were partly based upon the examination of cases clinically diagnosed general paralysis, which later experience taught him to exclude from that group, and which at the present day would not even clinically be considered as clear cases of general paralysis.

Within the last decade the question of general paralysis has entered on a new phase; new methods and results have largely altered the formulation of the problems, and in the rapid transformation thus brought about adjustment to the altered situation has not always been adequate. It is hoped that in this communication the present situation of the problem may be brought to a clear focus; the want of a clear definition of the general problem hampers considerably a common understanding and stands in the way of useful coöperation. It is this want of a common understanding, and the facts which are responsible for it, that make it necessary to publish in some detail the observations of the cases which form the material of this thesis.

As this study is not historical in character, little reference will be made to the individual steps in the development of the three main lines of recent advance in our knowledge of general paralysis. In the clinical sphere the examination of the cerebro-spinal fluid has been the main acquisition; histopathological research has yielded valuable differential criteria; serological studies have established on firmer ground the connection between syphilis and general paralysis.*

The introduction of the cytological examination of the cerebro-spinal fluid into psychiatric procedure by the French school, in 1901, gave the clinician an extremely useful aid in the task of distinguishing between the organic and the functional psychoses, and made possible a more

* Vid. foot note p. 8.

refined diagnosis of the organic dementias; the method has proved to be of special value in differentiating general paralysis from certain similar clinical pictures, arising on a different anatomical basis (alcoholic dementia, arteriosclerotic dementia, senile dementia).

Where the practical problem arises in the individual case of differentiating between general paralysis and one of these latter forms of dementia, the cytological examination of the cerebro-spinal fluid is now an indispensable procedure; to report cases of somewhat doubtful symptomatology, e. g., presenting a picture similar to that of Korsakow's psychosis, without the results of lumbar puncture, and then to refer to these cases as examples of general paralysis is to needlessly cumber the literature. It is obvious that (in the absence of histopathological examination) this consideration should make one accept with reserve conclusions based on observations which antedate 1901.

In the histopathology of general paralysis the researches of Nissl[1] and Alzheimer,[2] published in 1904, make an epoch. For the first time Alzheimer gave a full description of the histopathological changes in general paralysis on the basis of an adequate material (170 cases), his communication being admirably illustrated; since 1904, therefore, we have had available a trustworthy histopathological criterion of general paralysis, which has superseded the naked eye appearances of the brain and the merely impressionistic result of microscopical examination.

It is important to receive with caution results based on cases where this criterion has not been applied, for naked eye appearances may be inconclusive and a microscopical examination, which is expressed in such terms as, e. g., "round-celled infiltrate," embracing glia-cells, lymphocytes, plasma-cells, indicates an altogether insufficient analysis.

Alzheimer cites examples to show how misleading gross appearances may be; in a case, clinically diagnosed senile dementia, the autopsy disclosed thickening of the cranium with disappearance of the diploe, very pronounced thickening and clouding of the pia, hydrocephalus externus and internus, ependymal granulations, marked cerebral

atrophy, most marked in the frontal region; the microscopical examination, however, confirmed the clinical diagnosis. In another case the microscopical examination confirmed the clinical diagnosis of general paralysis, although at the autopsy there was nothing in the gross appearance of the brain to suggest the diagnosis.

An excellent example of the danger of making a diagnosis on the basis of the naked eye appearance of the brain, in a clinically doubtful case, is furnished by Case 5 of the present series, where the naked eye appearances led the pathologist, who did the autopsy, to consider the case one of cerebral arteriosclerosis and to rule out general paralysis; microscopical examination, however, disclosed the histopathological changes of general paralysis. The case had clinically presented many difficulties; without a satisfactory microscopical examination this case might have been used as a case of arteriosclerotic dementia to invalidate the differential importance of certain of the clinical features (the disorder of speech and of writing, the pupillary anomalies, the character of the attacks, the progressive course). Such a case shows the danger of using observations of older date, even although anatomical examination is said to have confirmed the clinical diagnosis. To a certain extent, therefore, the whole subject of general paralysis needs to be worked over anew; and the presentation of the case-material used in this thesis can not be considered superfluous.

The third great advance in the last decade was the introduction in 1906 of the method of Wassermann and its application to the problem of general paralysis; there was thus given a bio-chemical demonstration of the connection of syphilis with general paralysis, which had hitherto rested mainly on statistical grounds.*

In none of the three lines of advance has finality been reached; each step in advance has raised wider problems. The cytological examination of the cerebro-spinal fluid has enabled one to recognize many cases of general paralysis

*Since this was written in 1911 the last link in the chain of evidence establishing the syphilitic etiology of general paralysis has been supplied by H. Noguchi and J. W. Moore: A Demonstration of Treponema Pallidum in the Brain in Cases of General Paralysis. Jour. Exper. Med., Feb., 1913.

The term meta-syphilis will have to yield to that of parenchymatous syphilis.

which previously escaped attention, and to exclude many cases of dementia which had previously been wrongly included in the group of general paralysis. The meaning of a lymphocytosis of the cerebro-spinal fluid is, however, still a matter of considerable obscurity; it may be met with in a great variety of conditions, e. g., in cerebral symphilis, in tabes, in non-syphilitic brain tumor, in multiple sclerosis, perhaps in cerebral arteriosclerosis with cortical softening, etc. In no case can the diagnosis be mechanically turned out from the centrifuge.

The exact value of a lymphocytosis with regard to the diagnosis of general paralysis is not definitely settled, and the question is needlessly complicated by observations of doubtful value which are found in the literature; thus cases of general paralysis without lymphocytosis are referred to, even although there has been no anatomical examination. In this connection it must be remembered that the determination of the presence or absence of a lymphocytosis depends upon a somewhat delicate technique (if one use the French method); the absence of lymphocytosis in a suspected case of general paralysis should call for a renewed examination of the cerebro-spinal fluid, and for a more searching clinical examination, while the case can only have demonstrative value when the clinical diagnosis has been confirmed by autopsy with microscopical examination.

The researches of Nissl and Alzheimer have not finally solved the problem of the essence of the paralytic process; they have, however, yielded an invaluable practical criterion. That this is not the essence of the process may be seen by the fact that the same histopathological changes were found by Nissl in the cortex of a dog and of two rabbits. The great advance made by the publication of Nissl and Alzheimer is that it enables us in the whole general paralysis question to start from a homogeneous group, with regard to which a common understanding can be arrived at. To start from this common ground, to restrict the term general paralysis to this central homogeneous group is not to beg the question, nor to prejudge the question of the actual relationship of certain difficult cases to the classical

type of general paralysis. The clinical and anatomical conception of general paralysis may in the course of further investigations be further extended and modified; from the point of view of method the only safe starting point is that which limits the term to a certain clinical group, the limits of which are defined provisionally by the histopathological criteria of Nissl and Alzheimer. That this attitude needs to be emphasized is apparent on reading the observations in the literature; even such a serious author as Nonne,[3] in discussing the difficult question of the curability of general paralysis, bases his view on four observations, the propriety of which must be questioned, especially when one is aware of the extraordinary difficulty of differentiating clinically between cerebral syphilis and general paralysis. The question of method is here so important, that these cases may be referred to in detail. In the first case there is no mention made of an autopsy; the case as reported can not be differentiated from one of cerebral syphilis and the date of the observation is not given. This is important as, during the last ten years, the analysis of the clinical picture has received much more attention than previously, and clinical differentiation has made considerable progress. Nonne's first case is not clearer clinically than that of the syphilitic wood-carver Gennaro P. (to be discussed later in the remarks on Case 1); but the point to be emphasized in the latter case is that we are not entitled at the present date to make a positive diagnosis on the clinical grounds alone, even after a cytological and serological examination of the cerebro-spinal fluid. The second case of Nonne suggests as much the clinical picture of cerebral syphilis (endarteritis and meningitis) as that of general paralysis; the course tends to confirm the former diagnosis; without histopathological evidence the diagnosis of general paralysis in this case is far from established. In the third case the mental picture receives very summary mention, the physical picture is inconclusive, no autopsy was made; Case 4 of my series (W. C.) presented a clinical picture equally suggestive of general paralysis, but anatomical examination disclosed the presence of gummata in the brain. In Nonne's

fourth case the diagnosis of cerebral syphilis can not be excluded, and, to judge by the summary report and the course of the disease, seems as probable as that of general paralysis.

These cases have been referred to in some detail in order that it may be fully realized how current views on general paralysis are apt to be based on very unsatisfactory evidence, and how even serious workers fail to realize the difficulty of the clinical differentiation of cerebral syphilis and general paralysis.

In many reports of atypical cases we read the brief statement that the case was diagnosed general paralysis by a "competent authority"; such a statement has no value in comparison with a presentation of the clinical facts with, where possible, the results of a histopathological examination. The clinical differentiation of cerebral syphilis from general paralysis is in certain cases at present *impossible*, and even the histopathological differentiation may occasionally present the greatest difficulty; the pathologist has not said the last word on the question.

With regard to such questions as whether general paralysis is curable, general paralysis is at present in the same position as tuberculous meningitis was before lumbar puncture gave one an absolute clinical criterion of the disease. Osler in the third edition of his text-book (1898), discussing the prognosis of tuberculous meningitis, says, "Cases of recovery have been reported by reliable authorities, but they are extremely rare, and there is always a reasonable doubt as to the correctness of the diagnosis."

Since then a few cases of recovery of absolutely certain diagnosis (demonstration of the bacillus in the cerebrospinal fluid) have been placed on record. What have we in general paralysis to compare with the demonstration of the bacillus in tuberculous meningitis?

The most recent of the three important advances in the study of general paralysis has been the introduction of the Wassermann reaction in 1906. This reaction, elaborated in accordance with certain serological principles, is a complicated biochemical process, the interpretation of which is not

free from ambiguity, however valuable it is as an empirical aid in differentiation. Its exact value as an aid towards differentiating a syphilitic from a metasyphilitic process in the individual case is not yet definitely established. While the value of the reaction itself is under discussion, it can not be used to decide definitely a difficult individual case, otherwise there is apt to be a vicious circle; the reaction is used to classify the cases, and then the cases are used to determine the value of the reaction. In the absence of an unexceptionable clinical criterion cases of atypical symptomatology or course should not be used in determining the differential value of the reaction, unless there has been also a histopathological examination.

I have recently had under observation a woman (I. T.) who, on her first admission to the hospital in March, 1899, was diagnosed general paralysis on what appeared to be satisfactory grounds; after ten months she was discharged improved and was able to conduct her household, except during two brief periods, for the following ten years. On re-admission in December, 1909, she presented a characteristic picture of the simple demented type of general paralysis, with characteristic defect of memory; the knee-jerks were active, the pupils were unequal, reacted very faintly to light, defectively on accommodation; tremor of tongue; the speech was hesitating without tremor or transposition of letters; the writing showed striking distortion of words with transposition of letters; slight lymphocytosis of the cerebro-spinal fluid, 4 to 8 cells in the oil-immersion field, with positive butyric acid test; the cerebro-spinal fluid, examined by Dr. Noguchi by the original Wassermann method and by his own modification, gave a negative reaction. In view of the atypical course of the disorder I do not consider that it is justifiable to use such a case as an example of general paralysis in a discussion of the value of the Wassermann reaction, until the clinical diagnosis, however well founded it may appear, is confirmed by microscopical examination.

That the results of the cytological and serological examination of the cerebro-spinal fluid require to be carefully weighed along with the history of the development and the

symptomatology, in the formulation of a diagnosis, is obvious from the following case. The patient (H. W.), a man of 40 with a history of syphilitic infection, was admitted in a state of mild euphoric dementia; there was double optic atrophy, exaggeration of the knee-jerks, slight tremor of the hands and tongue, practically intact speech; no history of vomiting nor of headache; the cerebro-spinal fluid contained 110 cells per c. mm., and gave positive reaction with the Noguchi modification of the Wassermann method, as did the blood serum (Dr. Henderson).

In the absence of tumor symptoms the diagnosis of general paralysis was at first considered to be the most probable; the further course was a progressive decline without additional neurological symptoms; it was therefore thought that, in the absence of the more distinctive clinical features of general paralysis, a basal process of syphilitic nature (gumma, meningitis) was more probable. The autopsy disclosed an endothelioma in the region of the hypophysis, without any evidence of general paralysis or of a syphilitic process. The serological examination in this case had been rather misleading.

It is hoped that the preceding argument is sufficient to demonstrate the cardinal importance of the rôle played by histopathological examination in the prosecution of researches on general paralysis.

The following is a brief summary of the histopathological changes characteristic of general paralysis, as described by Alzheimer in 1904.

1. The *pia mater* shows diffuse changes, usually most marked over the frontal lobe; these changes consist essentially of an infiltration of the pia with cellular elements, plasma-cells, lymphocytes and mast-cells; in addition the vessel-walls show progressive and regressive changes.

With regard to the cortex it is convenient to describe first the mesodermal elements and then the ectodermal elements.

2. There is proliferation of the endothelial cells of the vessels with a marked tendency to the new formation of vessels through sprouting and vascularization of the proliferated intima. There is increase of the elastica and pro-

liferation of the adventitia; there is widening and infiltration of the lymph-spaces, which exist in the adventitial coat of the vessel wall. Among the infiltrating cells, plasma-cells are the most numerous; they are never absent in a case of general paralysis, even in the most acute. Lymphocytes and mast-cells are also found in the infiltrate. In advanced cases the vessel walls show regressive changes. Long rod-shaped or sausage-shaped cells are found in the cortex, their long diameter tending to run parallel with the medullary rays.

As to the ectodermal elements:

3. The nerve-cells show a great variety of degenerative forms, the meaning of which is as yet quite obscure; in advanced cases the nerve cells have in part disappeared. The usual orderly arrangement of the cells in the cortex is more or less disturbed. There is usually considerable degeneration of the medullated fibres in the cortex.

With regard to the non-nervous ectodermal tisue, *i. e.*, the neuroglia:

4. There is always a marked proliferation of the glia; this proliferation leads at first to the formation of numerous large glia cells, which form a large number of fibers and in very advanced cases dense tissue of thick glia fibers. The most marked increase is situated in the molecular layer and along the vessel-sheaths.

The changes in the rest of the nervous system in general paralysis, the nature and degree of affection of the central ganglia, cerebellum, spinal cord, need not be referred to here.

The work of Nissl and Alzheimer has not solved all the problems of the pathological anatomy of general paralysis; the meaning of the various elements in the histopathological picture, and the relation of the whole picture to the disease itself are quite obscure; the exact origin of various cells is still far from clear; the interpretation of certain findings has required revision, e. g., certain vessel changes, considered by Alzheimer to represent a progressive proliferative process, have been shown by Cerletti,[4] using new technique, to be due probably to a regressive process. The

value of the hispathological criterion, however, has in no way been affected by such minor modifications, and it enables us, in taking up the study of the organic dementias, to start with a common understanding in a way which was impossible before 1904.

So long as there was no definite criterion of what was general paralysis, no clear delimitation of the symptomatology was possible, nor could the wider problems of the disorder be satisfactorily attacked. In this communication, which is offered as a contribution to the symptomatology and pathological anatomy of the disorder, an endeavor is made to focus as clearly as possible certain central problems of general paralysis and to present some clinical material which may help towards the solution of these problems; to understand clearly the nature of a problem is already a step towards its solution.

The symptomatology of general paralysis is so varied that, in discussing the differential diagnosis, almost every other form of mental disorder must be referred to. The difficulty of diagnosis is not only present in the incipient stage of the disorder; even in the later stages, when the patient presents symptoms of advanced physical and mental deterioration, there may be great difficulty in distinguishing between general paralysis and other organic dementias. In the incipient stage the problem usually consists in differentiating general paralysis from such conditions as neurasthenia, a functional depression or excitement (melancholia, mania, manic-depressive insanity), a toxic or infective-exhaustive psychosis, etc. The introduction of the examination of the cerebro-spinal fluid has given invaluable aid in such questions of differential diagnosis. The problems presented by cases in which such difficulties arise are numerous; the rôle played by the constitution of the individual, the etiological importance of other factors than syphilis, the relation of the onset of the disorder to the type and date of syphilitic infection, and to its treatment, the factors which determine remissions or a course otherwise atypical, these are merely a few of the questions which have yet to be answered. In addition the meaning of the

individual symptoms, e. g., the sign of Argyll Robertson, the characteristic speech defect, etc., has yet to be cleared up. In the later stages of the disease the differentiation between general paralysis and cases of cerebral lues, alcoholic dementia, senile dementia, arteriosclerotic dementia, brain tumor, etc., is frequently difficult.

An important group consists of those patients, who present focal as well as general symptoms. The relation of the focal symptoms to the general disorder is variable; they may occur long before the onset of the general symptoms, they may come on simultaneously with the latter, they may develop when the general disorder has already reached a late stage. The relation of the focal symptoms to the general disorder may be more or less close; it may be a question of casual coincidence, as in certain cases of tumor and general paralysis, trauma and general paralysis; the relation may be close and lie in the fact that the cause of the focal symptoms has the same root as the process of general paralysis, as in the combination of endarteritis obliterans with general paralysis; the focal symptoms may be still more closely related to the general disorder and may be due to the localized severity of the process of general paralysis itself. A systematic study of this group of cases appeared to be desirable in order that somewhat loose current views on the nature of the focal symptoms in general paralysis might be replaced by more accurate views, based on material, in which both the clinical and the anatomical examination was adequate; the study of those cases, where the focal symptoms were not the direct expression of the paralytic process itself, promised to throw light on the actual evolution of the disease. The present communication embodies the results of such a study. In presenting these results the aim has been, from the clinical standpoint, to give clearer definition to the symptomatology and course of such disorders, rather than to undertake a detailed analysis of individual symptoms; and secondly, from the point of view of pathological anatomy, to give succinctly the main results, which allow at least a crude clinico-anatomical correlation and which have to be con-

sidered in forming a conception of the evolution of the disease; practically no reference will be made to the detailed histopathological studies made in these cases.

This communication, therefore, is more or less of the nature of a preliminary study, in which the main outlines of the problems are given, and the lines of further research indicated.

I have been compelled, with some regret, to relegate to an appendix the actual clinical observations, which have been reduced to as brief a form as is compatible with the purpose of this thesis. No one, who is familiar with the issues and appreciates the difficulty of mutual understanding, can fail to realize the inadequacy of a large proportion of the observations published in the existing literature. I have, therefore, given each observation in sufficient detail to enable the reader to form a personal opinion on the case; some of the cases, with extremely complex symptomatology and course, are unusually long. In order that the continuity of the presentation might not be interrupted by such long individual observations, the latter have been placed in an appendix; these observations, however, are considered to be of primary importance and to form the essence of the communication, and the brief summaries given in the text are inadequate substitutes for the observations in the appendix.

The material used in this communication consists of 19 cases of general paralysis with focal symptoms, which were personally observed in the clinical service of the Psychiatric Institute of the New York State Hospitals, and in which a systematic examination of the central nervous system was made post mortem. The observations began in 1905 when the male division of the clinical service of the Psychiatric Institute was organized; several of the cases had already been in the hospital for some time; Case 14 (M. H.) was worked up by Dr. G. H. Kirby and was personally observed during the later period of her illness; Case 10 (G. W.) was under the care of Dr. D. K. Henderson. The autopsies were performed by Dr. G. Y. Rusk, with the exception of one of the later cases which was done by Dr. C. I. Lambert;

Dr. Lambert also studied the condition of the vessels in several of the cases, and I am indebted to him for several of the photographs illustrating the pathological changes in the vessels. All the technical work connected with the preparation of the brains was done in the laboratory of the Psychiatric Institute under the direction of Dr. C. B. Dunlap, Chief Associate in Neuropathology. Dr. Dunlap also made a careful routine examination of each brain and confirmed the diagnosis; some of the material was used by him for special topographical studies; his reports on these brains were placed freely at my disposal. I owe Dr. Dunlap a special debt of gratitude for his coöperation during the whole course of this work and for his constant willingness to discuss the interpretation of the pathological findings. The clinical study of the cases was carried on with the constant advice and encouragement of Dr. Adolf Meyer, then Director of the Psychiatric Institute, to whom in this, as in other studies, I owe so much.

I take this opportunity of thanking Dr. August Hoch, Director of the Psychiatric Institute, for his kindness in making this publication possible and in going over the communication with a view to its publication.

A few remarks as to the extent and methods of examination of the nervous system may here be in place. In every case a careful surface examination of the brain and its membranes was made, and microscopical sections from representative areas confirmed the clinical diagnosis; in the majority of the cases the fore-brain, mid-brain and hind-brain were cut in serial slices of less than 1 cm. thick so that no focus of softening was likely to escape detection. In the few cases where this was not done owing to preparations of a special nature being desired, an equally thorough search for focal softenings was made. Further study of the brain was carried on according to the type of lesion involved; in cases with vascular disorder and focal lesions the vessels received special attention and were studied in serial sections; in some cases with focal lesions the whole brain was cut in serial sections. Where the clinical symptoms indicated the likelihood of a specially localized severity

of the cortical process large topographical slices were cut, in order that the severity of the cortical process in various areas might be compared. The desirability of such cortical studies was emphasized by Nissl, but certain technical difficulties have to be considered. It is practically impossible to make a thorough cortical study of a whole brain. Alzheimer followed the method of examining small blocks from the various areas. It is obvious that this method leaves much to be desired, as so much territory remains unexplored. On the other hand the use of very large sections presents drawbacks; large celloidin sections are too thick for fine histological studies, and large paraffin sections, even although cut at 10 $\mu$ by the special microtome in use at the Institute, do not stain so satisfactorily as the smaller sections. The large paraffin sections, however, are adequate for certain topographical studies.

The difficulties in the interpretation of topographical variations are considerable. It must be remembered that the histopathological features which are characteristic of the paralytic cortex are merely reliable diagnostic criteria of the morbid process; we are not entitled to assume that the degree of intensity of these changes is a reliable guide to the severity of the morbid process, still less to the severity of the process at an earlier date. Moreover the various elements in the histopathological picture—vascular changes, perivascular infiltrate, degeneration of nerve-cells, neuroglia changes—do not always vary *pari passu*, and it must be remembered that we can not satisfactorily demonstrate an œdema or non-vascular exudate. We have therefore no sound standard of measurement of the severity of the process in any particular cortical area, and can at best only give an impressionistic opinion.

The 19 cases used in this study have been classified in the following four groups:

GROUP 1: *Cases of general paralysis with focal symptoms on the basis of vascular disorders* (4 cases). Along with these cases is presented a case of brain syphilis (gumma), in which the clinical picture could not be differentiated from that of general paralysis.

GROUP 2: *Cases of general paralysis with focal symptoms on the basis of localized severity of the process of general paralysis; Lissauer's Atypical General Paralysis* (6 cases).

GROUP 3: *Cases of general paralysis with focal symptoms of traumatic origin* (2 cases).

GROUP 4: *Cases of general paralysis with focal symptoms which are correlated neither with vascular disorders nor with localized severity of the process of general paralysis* (6 cases).

GROUP I. *Cases of general paralysis with focal symptoms on the basis of vascular disorders.*

Case 1. W. M.
Case 2. T. R.
Case 3. J. L. W.
Case 5. M. D. R.
Case 4. W. C. (Lues cerebri).

CASE 1 (p. 63). W. M., born in 1861; moderately alcoholic; no definite history of syphilis; twice married; wife never pregnant; in 1894 sudden onset of weakness of left arm and leg (? face) without unconsciousness; very slight residual weakness; in 1902–03, insidious onset of classical general paralysis; November 18, 1905, general convulsions followed by twitching and limpness of the left arm and leg, and by quasi-purposeful movements of the right arm; May 7, 1906, general convulsions with special twitching of the left face, arm and leg, followed by the same quasi-purposeful movements of the right arm; November 30, 1906, death.

*Pathological Anatomy.* Cortex, typical changes of general paralysis; subcortical softening of R. F. 3 (vid. Fig. 1); endarteritis obliterans, aneurysmal dilatation and occlusion of cerebral vessels (vid. Fig. 2 A).

*Remarks on Case 1.* In this case the first neurological episode consisted of a hemiplegic attack at the age of 33. At that age, in the absence of any general infection or valvular heart lesion, and of a few conditions such as brain tumor, a hemiplegic attack may safely be attributed to a syphilitic disorder, most commonly a syphilitic endarteritis. Eight years later the patient began to present evidence of

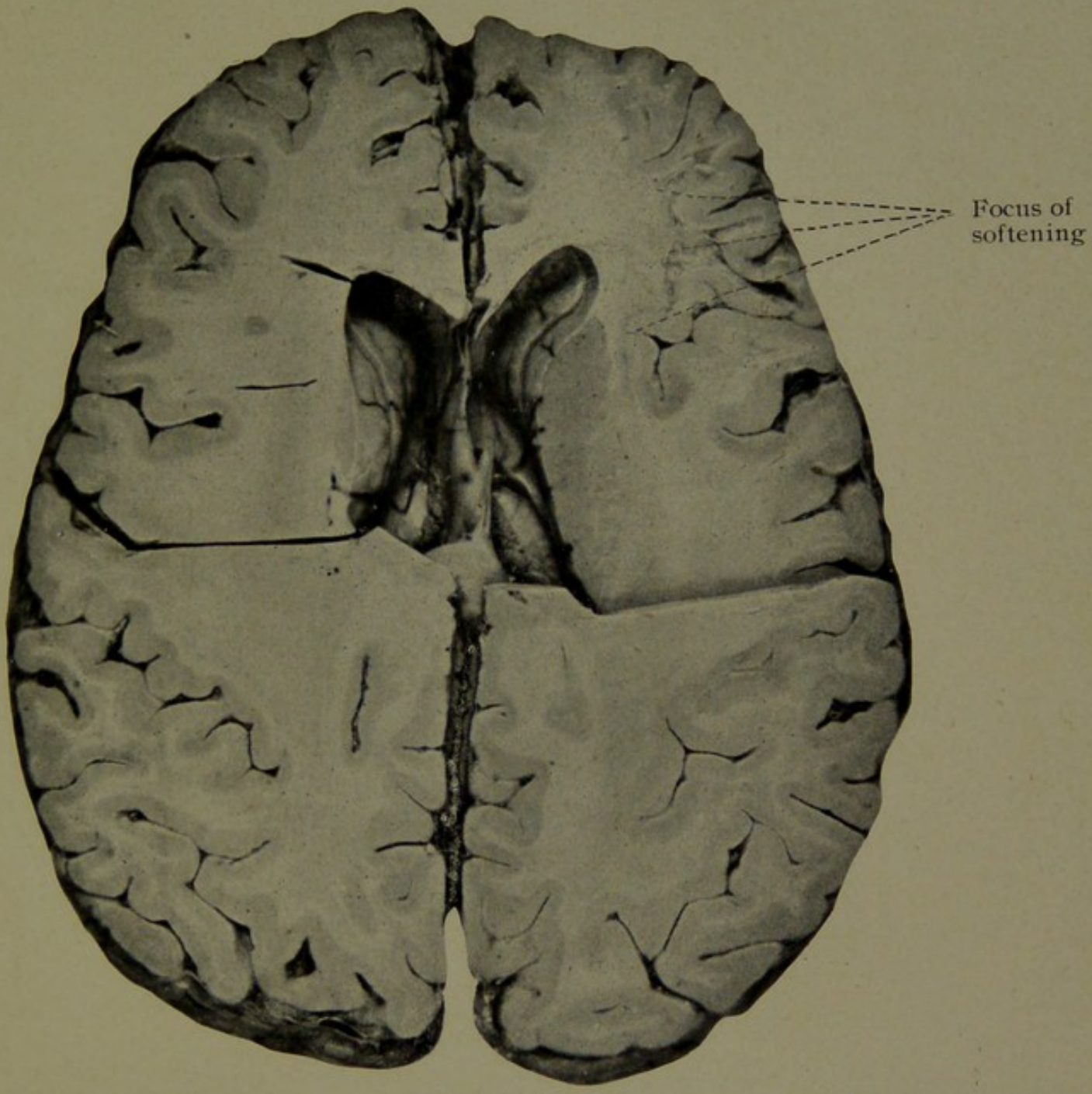



Fig. 1

Case 1 (W. M.) Sub-cortical softening in R. F.$_3$

general paralysis. We, therefore, have here to deal with the insidious evolution of a case of syphilitic cerebral vascular disease into a case of general paralysis. In view of the fact that general paralysis seems only to occur in patients, who have at a long antecedent date had syphilis, it is not at all to be wondered at that at an earlier period the brain should be affected by the syphilitic poison. The fact that a brain at a late date is going to succumb to general paralysis is no guarantee of immunity from the earlier syphilitic lesions. The exact period of the evolution when the process has ceased to be merely a syphilitic process, and when the morbid process of general paralysis begins, and the nature of this evolution are questions of great interest. They raise the further question whether in the ordinary case of general paralysis the process is a unitary one, or whether there may not be at the same time both a syphilitic element and a meta-syphilitic element in the picture, or whether after all the so-called meta-syphilitic element may not merely be the further evolution of the syphilitic process. There are cases of brain syphilis, in which, alongside of the definite syphilitic lesions, there are histopathological changes so closely similar to those of general paralysis that only a careful analysis enables the distinction to be made; there can be no reasonable doubt that such cases are frequently, perhaps, as a rule, considered to be cases of general paralysis. Thus in the case of W. C. (Case 4, to be referred to later) in addition to gummata there was present a type of syphilitic disorder, to which Dunlap[6] has recently called attention, which so closely resembles the process of general paralysis that the two are with difficulty separated. On the other hand Nissl has called attention to the fact that the proliferative changes in the cortical vessels in general paralysis are quite similar to those met with in a type of syphilitic vascular cortical disorder without any cellular exudate, which is associated with his name (syphilitic endarteritis of the small cortical vessels, Nissl); he has suggested the possibility that these vascular changes in general paralysis may have to be considered as directly syphilitic and that a

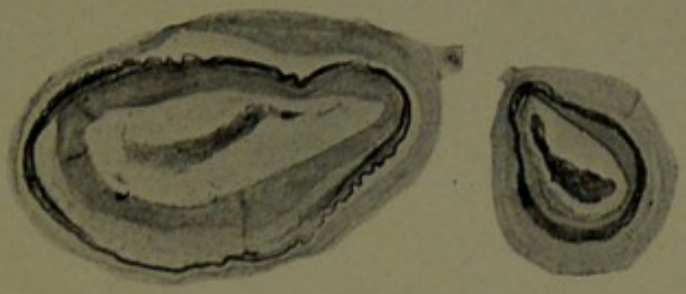
similar explanation for the degenerative processes of the parenchymatous elements can not as yet be excluded. The limits of our knowledge of the actual evolution of the process of general paralysis is further illustrated by a case to which brief reference may be made.

Gennaro P., had syphilis at the age of 19; in December, 1907, at the age of 40, he had an apoplectiform attack with residual left-sided weakness; after this attack he was inefficient at work, treated his wife outrageously, and finally was certified as insane. Physical status on admission (May 19, 1910): slight weakness of the left side, pupils Argyll Robertson, no defect of speech nor of writing, no tremor of fingers; lymphocytosis and increased globulin content of the cerebro-spinal fluid, Wassermann (Noguchi modification) positive with the cerebro-spinal fluid and blood serum (Dr. Henderson); the mental state was one of mild complacency with inadequate realization of the situation; his memory was slightly defective.

In this case we attribute the hemiplegic syndrome to a syphilitic endarteritis; the Argyll Robertson pupils and other symptoms point in the direction of general paralysis. The Wassermann reaction, which is so useful as a diagnostic criterion in separating syphilitic and non-syphilitic disorders here leaves us in the lurch; statistically, a positive reaction points towards general paralysis, but, in the present state of our knowledge, the further course of this case and of similar cases must be looked to for help in giving to the reaction its exact significance, while the reaction does not as yet enable us to form a more definite conception of the individual case. The reaction although elaborated according to certain biochemical principles is essentially an empirical criterion, and throws as yet comparatively little light on the actual evolution of the disease.

Case 2 (p. 66). T. R., born in 1863, right-handed; syphilis (? date); February, 1897, transitory weakness of the right hand and impairment of speech, without loss of consciousness; November, 1897, attack of weakness of left arm and leg with impaired speech, without loss of consciousness; mental symptoms for two weeks; permanent residual

A



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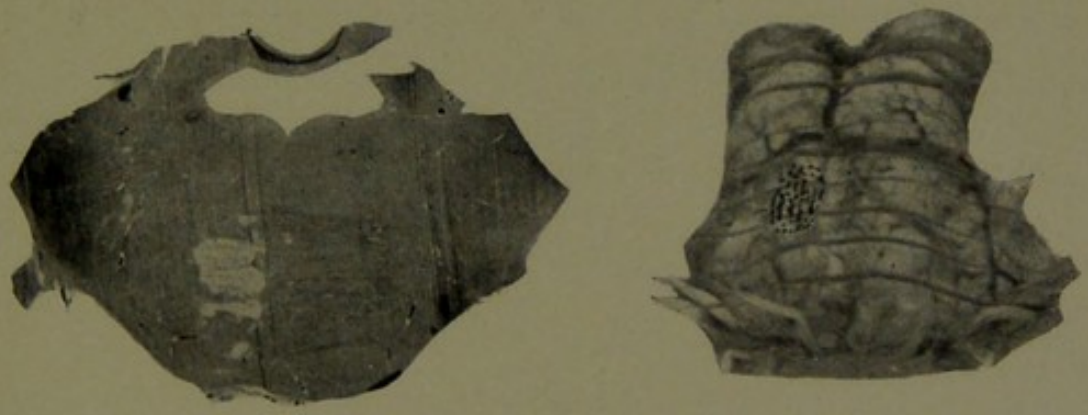


Fig. 2

A) Case 1 (W. M.) Microphotograph of the right middle cerebral artery and the branch supplying the area of softening.

B) Case 2 (T. R.) Photograph of the pons through the area of softening, and photograph of the mid-brain and pons to show the site of the softening.

weakness of left leg, after several weeks no weakness of left arm; during the following 9 years episodes of weakness with exacerbation of limp; 1906, onset of classical general paralysis; rapid deterioration.

April 13, 1907, exacerbation of weakness of left leg; April 14, attack of unconsciousness, coma; death on April 16.

*Pathological Anatomy.* Cortex, typical changes of general paralysis; softening in the right side of the pons (vid. Fig. 2-B.); endarteritis obliterans.

*Remarks on Case 2.* The general course of the disorder in this case was essentially parallel to that of Case 1 (W. M.); the duration of the interval between the first hemiplegic attack and the insidious onset of the general paralysis was almost the same. The seat of the focal lesion was only discovered after a thorough examination of the brain, and the case shows the necessity of an examination being complete before any deductions as to clinico-anatomical correlation are drawn. The vascular change responsible for the focal softening was endarteritis obliterans (Heubner) so that here too the clinical picture of the case was modified by a syphilitic lesion.

An interesting clinical detail was the comparatively slight involvement of the arm with a lesion in the region shown in the accompanying photograph.

Case 3 (p. 68). J. L. W., born in 1863, right-handed; alcoholic; 1898, chancre with treatment of six months; August, 1901, diplopia, improving under potassium iodide; April, 1902, paralysis of left side, coming on in 24 hours, without loss of consciousness; improvement under treatment; November, 1902, paralysis of left side with clonic convulsions, and with inability to speak, without loss of consciousness; improvement under treatment; 1902–1905, occasional headache; autumn, 1905, inefficient; October, 1905, progressive weakness of the left leg, loss of memory; January, 1906, twice fell without loss of consciousness; January–August, 1906, vigorous antisyphilitic treatment without improvement; mental symptoms; February, 1906, four convulsions; August 14, series of convulsions, with special involvement of the right side, with residual weakness of right arm and

marked paraphasia; diminished sensibilty over right face and arm; no hemianopia but statement of less distinct vision in the right visual field; occasional twitching of right arm; death August 26, 1906.

*Pathological Anatomy.* Cortex, typical changes of general paralysis; focus of softening, of uncertain age, involving part of R. F. 1, of R. F. 2, and of upper fourth of R. A. C.; old focus of softening in the head of the left caudate nucleus and in the anterior fourth of the left putamen; focus of softening in the marrow of the left occipital lobe; foci of softening in the pons; small cortical wedges of atrophy of the ectodermal elements; endarteritis obliterans.

*Remarks on Case 3.* In this case the considerations, which have been discussed in regard to the two previous cases, assume still greater prominence; in the former cases the clinical history is divided into two very distinct periods, the symptomatology of each of which has a certain independence. In the earlier period the symptomatology is merely that of focal softening on the basis of vascular disease; in the later period the clinical picture is that of a classical general paralysis, with certain additional features referable to the already existing gross lesion, viz., the local distribution and character of the attacks. The relation of the process in the second period to that of the first period is not suggested immediately by the clinical history, but by general considerations on the etiology and nature of the two processes; apart from these considerations the supposition of two quite independent processes would be adequate. In Case 3, on the contrary, the clinical history does not divide itself into such distinct periods, but seems to present the evolution of one process, an evolution in which no definite line of demarcation can be drawn; the picture of brain syphilis passes insidiously and without any long intervening period of incubation into that of general paralysis. As sufficient evidence for this, it may be stated that the patient until one month before death was diagnosed and treated as a case of brain syphilis by a most competent neurologist.

The whole early course of the disorder, its early incidence

after the initial infection, the presence of certain symptoms, on which stress is frequently and justly laid as aids in the differentiation of brain syphilis from general paralysis, viz., diplopia, headache, good insight, absence of grandiose ideas, seemed to indicate brain syphilis; the euphoria shown by the patient is nothing unusual in the latter disorder, while the memory defect, in its setting of a certain mild confusion and difficulty of orientation, could not be considered as pathognomonic of general paralysis. At the beginning, therefore, the process appears to have been that of brain syphilis, as evidenced by the close relation to the initial infection, by the symptomatology, by the pathological evidence of focal lesions on a vascular basis; while at the end the process was undoubtedly that of general paralysis, as evidenced by the typical histopathological changes in the cortex.

The limits of our knowledge of the development of general paralysis are brought into clear relief by this case. We have not at the present day clinical criteria which enable us to say at what exact stage in the individual case the process is no longer merely a syphilitic one and that another and more serious process has begun to develop; when the latter process has developed to a certain extent, the clinical picture may leave us in no doubt, but we must clearly recognize the absence of any *clinical* criterion, which will give an answer as unequivocal as the only existing reliable criterion, viz., the histopathological picture. Even the Wassermann reaction leaves us in doubt, although it is one additional factor which has to be considered in the weighing of the facts in the individual case; the serologist, to whom the clinician has turned for some test of infallible accuracy, gives the clinician back the problem with the additional task of valuating the serological data.

This case, along with the following one, a case of brain syphilis, illustrates the great necessity of careful clinical differentiation, and the danger of using in any research on general paralysis or brain syphilis cases where histopathological examination is excluded, unless the limits of our clinical criteria are fully appreciated.

CASE 4 (p. 72). W. C., born in 1858; chancre 1891; very alcoholic, with long standing delusions of jealousy and of poison; summer, 1900, squint of several months duration; September, 1900, attack of dysarthria and staggering, with gradual improvement of speech; September, 1901, weakness of right leg, speech difficulty; residual weakness of right leg; 1901–1903, several attacks of weakness of the right side with involvement of speech; 1903, transitory weakness of left arm with inability to speak; July, 1905, attack of unconsciousness, followed by stuporous condition and delirium of several weeks duration; August, 1905, on admission, euphoric dementia; October 25, left-sided ptosis of four weeks duration; right-sided weakness, sign of Babinski and ankle clonus on both sides; left internal ophthalmoplegia, defective speech and writing, lymphocytosis of the cerebro-spinal fluid, optic discs normal; April 30, 1906, general convulsion after which the right arm was held rather rigid; death on May 3, 1906.

*Pathological Anatomy.* Cortex, preservation of the general architectonic, no diffuse plasma-cell infiltrate; general pial cloudiness, frontal atrophy, ventricular granulations; gummata, one in left centrum ovale, (vid. Fig. 3), another in the right parieto-occipital fissure; syphilitic meningitis, of varying grade, with slight extension into the cortex; old softening, of vascular origin, in the right internal capsule and thalamus, another in the left side of the hind brain involving the pyramidal fibres; endarteritis obliterans.

*Remarks on Case 4.* The difficulty of diagnosing clinically between cerebral syphilis and general paralysis is again strikingly shown by this case. The difficulty did not arise from extrinsic causes, such as a defective anamnesis, etc., nor from the fact that the clinical examination was not directed towards specific issues. The examination endeavored to define as clearly as possible the nature of the disorder of speech and writing, the type of memory defect, the degree of want of realization of the condition shown by the patient's mood and his plans for the future. The neurological incidents were in type very similar to those of the previous case; the left-sided ptosis and com-

Fig. 3

Case 4 (W. C.) Photograph showing gumma in the left centrum ovale. The connection with the pia of the insula is not shown in this section.

N. B. The photograph is taken of the under surface of the section.

plete fixity of the left pupil were the signs which most distinctly indicated a syphilitic process.

Even if the Wassermann reaction had at this date been introduced into psychiatric procedure, it is doubtful whether it would have thrown much light on the case; a negative reaction would have been of considerable weight, a positive reaction would have left the problem unchanged.

CASE 5 (p. 76). M. D. R., born in 1847; some venereal infection about 1870; about 1893 onset of memory defect; in 1902-'3 memory defect was pronounced, progressive; July, 1905, dazed episode, general convulsion; on admission (July 12, 1905) mild general mental reduction, poor memory and retention, poor orientation, no definite euphoria nor boastfulness; knee-jerks absent, pupillary reaction good, speech indistinct, not tremulous, writing tremulous and almost illegible, arteriosclerosis; September 3, 1905, two attacks of twitching most marked on the left side, without loss of consciousness; October 2, two general convulsions, followed by quasi-delirious behavior; November 5, general convulsion; November 21, series of convulsions of mild nature; November 22, twitching of arms; lymphocytosis of cerebrospinal fluid; during the following months occasional convulsions; March 23, 1906, left-sided attack, with weakness and ataxia of left arm, diminution of sensibility over the whole left side, left-sided hemianopia passing into complete blindness, duration of symptoms two days; April 3, during examination sudden pain in head, "black in part of the eyes;" April 4, right-sided hemianopia, confused semi-delirious remarks; clear after a few days; May 19, sudden blindness, eyes turned to the right, mild delirium; May 20, persistence of blindness in the right field; May 31, transitory loss of vision in the left field (*i. e.* complete blindness from double hemianopia), weakness of the left side (leg, arm), anesthesia of the whole left side; June 20, left-sided hemianopia (in addition to already existing right-sided hemianopia), twitching of the left face and arm; disappearance of the left-sided hemianopia in two days; July 15, left-sided hemianopia (causing complete blindness), twitching of left hand and face, left-sided

hemianesthesia; August 26, convulsion with transitory weakness of the left arm; September 10, attack of left-sided hemianopia with rigidity and twitching of the left arm; after three days clear vision in left field; October 12, temporary elimination of the left visual field, weakness of the left arm, impairment of sensibility on the whole left side; October 24, general convulsions beginning with twitching on the right side, leaving transitory impairment of sensibility in the left hand, and left-sided hemianopia; November 9, transitory left-sided hemianopia, twitching of the left face and platysma, left-sided anesthesia; after three days no residual; December 3, left-sided hemianopia, hemianesthesia, weakness of the hand; December 4, 5, series of convulsive attacks; December 5, left leg rigid, left-sided anesthesia, sign of Babinski on the right side; December 6, 7, continuation of twitching; December 7 to December 15, blind; December 31, peculiar movements of left and right arm of different type; January, 1907, mildly delirious, progressive impairment of pupillary reaction to light; March 2, twitching of right hand, arm and leg; March 25, twitching of right face, arm and leg, impaired sensibility on the right side; April, frequent episodes of clonic contractions especially of the right arm; for several weeks before death apparently blind; death on May 7, 1907.

*Pathological Anatomy.* Cortex, typical changes of general paralysis; in the left occipital lobe a large area of softening, involving the lips of the calcarine fissure (vide Fig. 4, 5); small softening in the left cuneus; atrophy of the right optic tract due to pressure of contiguous thickened vessels; old focus of softening in the left cerebellar lobe; marked atrophy of the medullary substance with occasional lacunae; marked thickening of the basal arteries, with secondary dilatations and tortuosity (vide Fig. 4), complete occlusion of the branch of the left posterior cerebral supplying the occipital focus of softening; slight degeneration of the posterior columns of the cord.

*Remarks on Case 5.* The patient was a man of 58, of poor physique and slightly defective mental development who was admitted in July, 1905, on account of an epilepti-

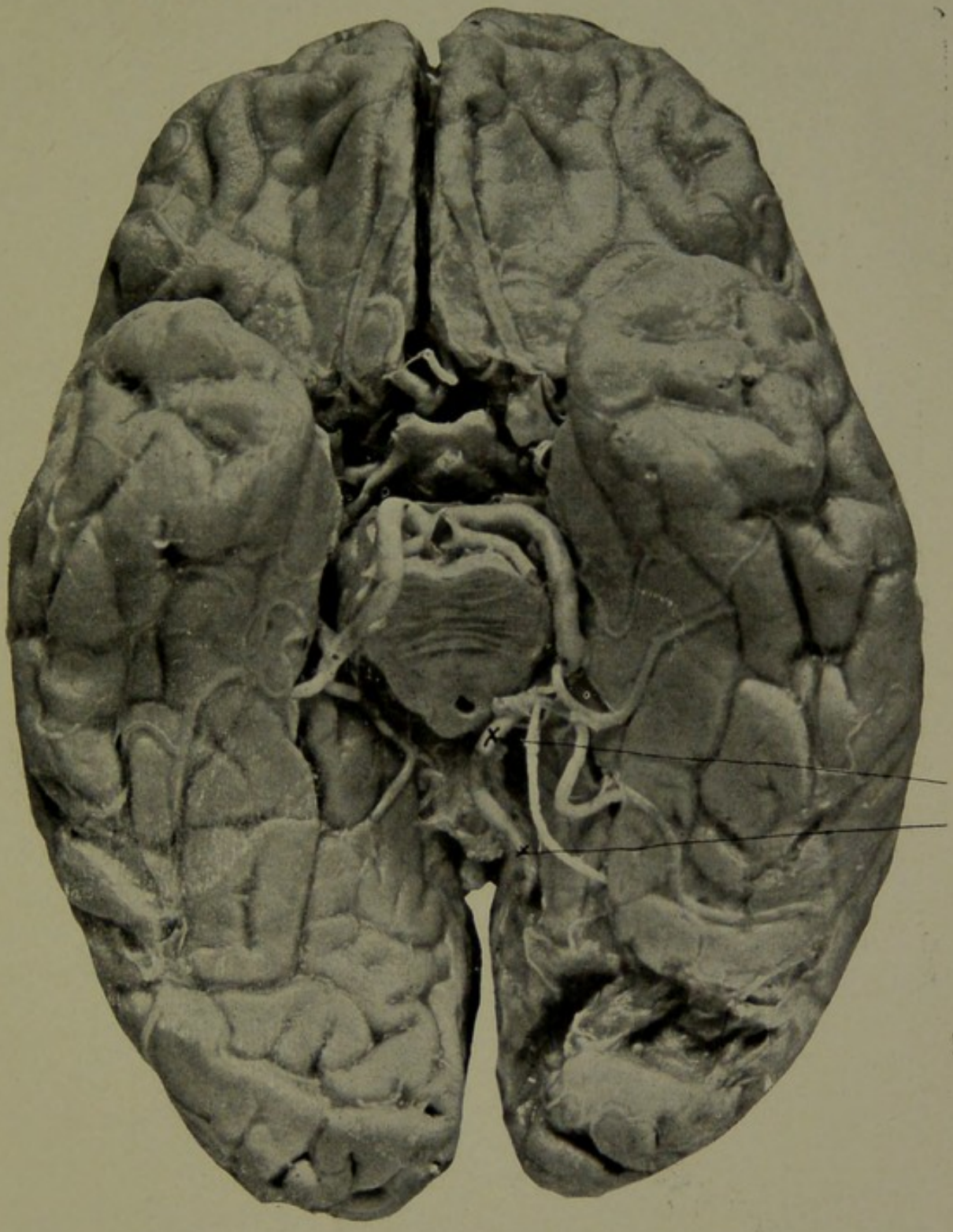


Fig. 4

Case 5 (M. D. R.) Photograph of the base of the brain after removal of the hind-brain. The right posterior cerebral artery has been pulled away from the optic tract, on which there is a well-marked groove. The focus of softening in the left occipital lobe is shown with the much thickened and occluded artery leading to it (calcarine artery).

A



B



Case 5 (M. D. R.) A) Photograph of a transverse section of the left occipital lobe showing extensive softening of the mesial aspect. At the right hand side of the section is seen a small cortical softening, magnified in the lower illustration (B). Note the preservation of the superficial layer of the cortex, the complete destruction of the ectodermal elements in the focus, the multitude of gitter cells. The softening is probably over a year old. Contrast this with the focal destruction of Case 13 (Fig. 9) where no gitter cells are present.

Fig. 5

form convulsion; this was his first convulsion. For over fourteen years his general efficiency had been diminishing, his memory especially had deteriorated; during the two years before admission his memory defect had become more accentuated.

Neither previous to admission nor during his stay in the hospital did the patient show any abnormal mental trend; his mental condition was similar to that of simple senile deterioration; he showed poor orientation especially for time, very marked memory defect especially for the recent past, some insight into his condition and fair preservation of his personality; his mood was one of simple good-humor, and he was at times mildly jocose. In connection with various attacks he was mildly delirious, but apart from these episodes his mental condition showed little change in hospital except progressive enfeeblement parallel with the physical decline, and a tendency towards the end to be in a dull hazy condition bordering on a delirium.

His physical condition on admission was that of a rather feeble old man with moderate arteriosclerosis; the knee-jerks were absent. During his residence in the hospital he had a series of localized and general convulsions, and developed a permanent right-sided hemianopia, while during the last weeks of life he was apparently blind.

The knee-jerks were absent but the patient did not present a frank tabetic complex; the pupils even at a late stage of the disorder showed only slightly defective reaction, the sign of Romberg was not present at an early stage, there was no localized sensory defect, no history of shooting pains. The posterior columns of the cord showed slight degeneration.

The patient had numerous convulsions, the severity of which was not uniform; in some the patient lost consciousness, in others he was delirious, in others he was in a confused state bordering on a delirium; the attacks were frequently accompanied by extreme distress and agitation, and sometimes by an agonizing headache. The most typical attacks showed, in addition to these general symp-

toms, the following transitory focal symptoms: (1) left-sided hemianopia, (2) paralytic motor symptoms on the left side, the arm being more affected than the leg, (3) irritative motor symptoms on the left side, with occasional involvement of the right, (4) left-sided anesthesia, the arm being most affected.

*Visual disorders* were obviously present in the earlier attacks; he was noted as "fumbling along the wall as if *groping* for imaginary objects." In the first attack observed in detail, along with the left-sided weakness there was left-sided hemianopia which during the examination passed into general blindness; there was no residual limitation of the visual field. On a later occasion he complained suddenly of headache, "I am black in part of the eyes" ("vision noire"). Next day he was completely blind in the forenoon; after this attack he had permanent right-sided hemianopia. In the various subsequent attacks the patient, in addition to the left-sided motor and sensory symptoms, had transitory left-sided hemianopia causing temporary blindness. The recovery from the left-sided visual defect was usually abrupt, but sometimes there was a transition period in which the patient had some visual perception of objects but was unable to name them.

*The motor phenomena* were irritative and paralytic. Both the irritative and the paralytic features were much more marked on the left side, and were in several attacks confined to that side. The attacks left no motor residual, but it was noted that the left side of the patient's face was somewhat flatter than the right. After one attack, which involved both sides, the sign of Babinski was permanently present on the right side.

The distribution of the *sensory disorder* varied from attack to attack, and in some the left arm alone was affected.

The combination of permanent right-sided symptoms (hemianopia, sign of Babinski) with left-sided attacks seemed to indicate a definite focus or foci of softening in the left hemisphere, with recurrent disorders, probably circulatory, in the right hemisphere, the visual sphere being

specially affected; it was thought probable that the basilar artery and posterior cerebral arteries presented an advanced degree of arteriosclerosis. The diagnosis of severe progressive arteriosclerotic brain degeneration was made, but in a paper written before the autopsy was made, the author said: "The absence of knee-jerks, sign of Romberg, marked lymphocytosis of the cerebro-spinal fluid, the sluggish, irregular pupils, force one to reserve the diagnosis; for it is not as yet sufficiently established what weight is to be laid on these signs, individually and collectively, in the clinical differentiation of the organic dementias."

The anatomical examination showed how complex the nature of the disorder actually was; without a satisfactory microscopical examination the case might, even after autopsy, have been considered as merely a case of arteriosclerotic brain degeneration, and not a case of general paralysis, and one more would have been added to the list of cases in the literature, which have done so much to confuse the whole problem of general paralysis.

The vessels, on microscopical examination, showed a very late stage of a girdling endarteritis with very marked degeneration of the thickened coats.

As to the exact meaning of the type of vascular disorder present in this case, a discussion of the point would lead one into very debatable ground and would raise the whole question of the pathology of arteriosclerosis; it is probable that the condition is essentially of syphilitic origin.

The syndrome—hemiplegia, hemianesthesia, hemianopia—if permanent, usually arises on the basis of a softening or hemorrhage, involving the internal capsule and the optic radiation. In Case 7, S. G., where this syndrome was present, the clinical syndrome could be crudely correlated with the extreme severity of the cortical disorder in the one hemisphere; in Case 17, A. H., neither a special localized severity of the cortical process nor a softening nor hemorrhage was demonstrated (the topographical study of the cortex was not sufficiently extensive to justify a final statement on the possibility of correlation in Case 17).

Where the syndrome is of brief duration, as in several of our cases, the supposition of a transitory ischemia, involving the relevant area of the white matter, may be entertained. The centripetal path, however, to the right visual cortex in the present case was vulnerable at another point, viz., where the right posterior cerebral artery pressed upon the optic tract posteriorly and where the internal carotid pressed upon the same tract anteriorly; here the tract had been reduced to a narrow ribbon. It is probable that many of the unusual features in regard to the hemianopic attacks, e. g., the seeing black in front of the eyes, the transition period of hazy vision as the hemianopia cleared up, were to be interpreted as symptoms of pressure on the optic tract.

For the explanation of the transitory motor and sensory symptoms, one is tempted simply to refer to the condition of the vessels; in view, however, of the cases described in our fourth group it is better to admit frankly that the exact mechanism of the attacks is obscure, and that we are not as yet in a condition to attribute its due weight to each of the two prominent elements demonstrable, the gross changes in the vessels and the morbid process in the cortex.

*Review of Group i.* The vessel changes in the four cases of general paralysis, included in this group, consisted either of a well-marked endarteritis obliterans, or of a less typical condition, complicated by considerable degeneration, which probably represented a late stage of the same disorder. The foci of softening produced by these vessel changes were situated in the cortex, the sub-cortex, the brain-marrow, the basal nuclei, the hind-brain. In addition to these typical softenings the thickened vessels had in one case at least caused direct damage by pressure.

The permanent focal symptoms in the first three cases consisted of hemiplegia, in the fourth case (*i. e.*, Case 5) of hemianopia.

The onset of the hemiplegia in these three cases was without loss of consciousness; it began in each case with a feeling of numbness or tingling in the hand, and it reached its maximum not abruptly, but within a period of 24 hours.

In none of the three cases was there any residual weakness of the face; in Case 2 the leg alone showed residual weakness. No residual sensory defect could be demonstrated in any of these cases.

In Case 2 no epileptiform attacks were observed, and the patient died after an apoplectiform attack. In the other three cases epileptiform convulsions, frequently of Jacksonian type, occurred. The epileptiform attacks were evidently somewhat modified by the existing gross lesions, e. g. in Case 1 twitching of the left (*i. e.* hemiplegic) side was observed as the first and also as the last feature of some of the attacks. The final episode in Case 3 was of the type more frequently met with in Group ii, that is a comparatively long period (two weeks) during which sporadic twitching occurred, with considerable variations in the mental level.

The symptomatology of Case 5 has already been discussed in some detail in the remarks on that case; no typical apoplectiform attacks were observed, but the attacks presented rather a mixture of the features of the apoplectiform attacks of the first three cases with the features of the epileptiform attacks of other cases (e. g. the attack on March 23, 1906). Such a mixture of features is probably the clinical expression of the complexity of the anatomical changes, where at the same time the vascular system is seriously impaired while the cortex is the seat of a progressive disorder of another type.

While the apoplectiform attacks in this case were to be interpreted in the light of the vascular disorders with the consequent focal softenings, Case 9 (J. D.) is a sufficient warning against making a diagnosis of "thrombosis of the internal capsule", or elsewhere, merely on the basis of an apoplectiform attack. In Case 9, where an apoplectiform attack left a residual hemiplegia, no focus of softening was discovered, and the vessels showed only moderate diffuse thickening.

The general bearing of Group i on the whole question of the relation of syphilis to general paralysis has been sufficiently discussed in the remarks on the individual cases.

GROUP II. *General paralysis with focal symptoms on the basis of localized severity of the general paralysis process:*

Case 6. S. G.
Case 7. R. F.
Case 8. A. S.
Case 9. J. D.
Case 10. G. W.
Case 11. M. T.

CASE 6 (p. 88). S. G., born in 1862; temperate; no definite history of syphilis; 1896, shooting pains, especially in the left leg; 1900, occasional numbness and weakness of the left leg; insidious change in disposition, progressive inefficiency; 1905, definite mental symptoms; June, 1906, on admission, classical general paralysis with tabes; June 4, 1907, general convulsion, followed by left-sided twitching of one week's duration, with residual left-sided weakness, impairment of sensibility, hemianopia; August 12, twitching of the left side of three days duration, at first confined to the leg and arm, then involving the thorax and neck, but not the face; later an occasional general convulsion; November 4, 1907, twitching of left face, arm, leg, with loss of consciousness, duration 1–½ minute; November 6, left-sided convulsion with increase of weakness of left side; November 30, 1907, onset of a series of general convulsions, with continuous left-sided twitching lasting one week; 1908, occasional attack; November 5, 1909, right-sided twitching with loss of consciousness; death on November 6, 1909.

*Pathological Anatomy.* Marked reduction of the whole right hemisphere, due to pronounced atrophy and shrivelling of the convolutions of all parts (vide Fig. 6); the upper third of the anterior central convolution and the base of the temporal lobe were somewhat less affected than the other areas; cortex, typical changes of general paralysis, more marked on the right than on the left side, the cortex on the right side being much narrower; no focal softening; slight thickening of the basal vessels, the right middle cerebral a little thicker than the left; degeneration of the posterior columns of the cord, and of the left crossed

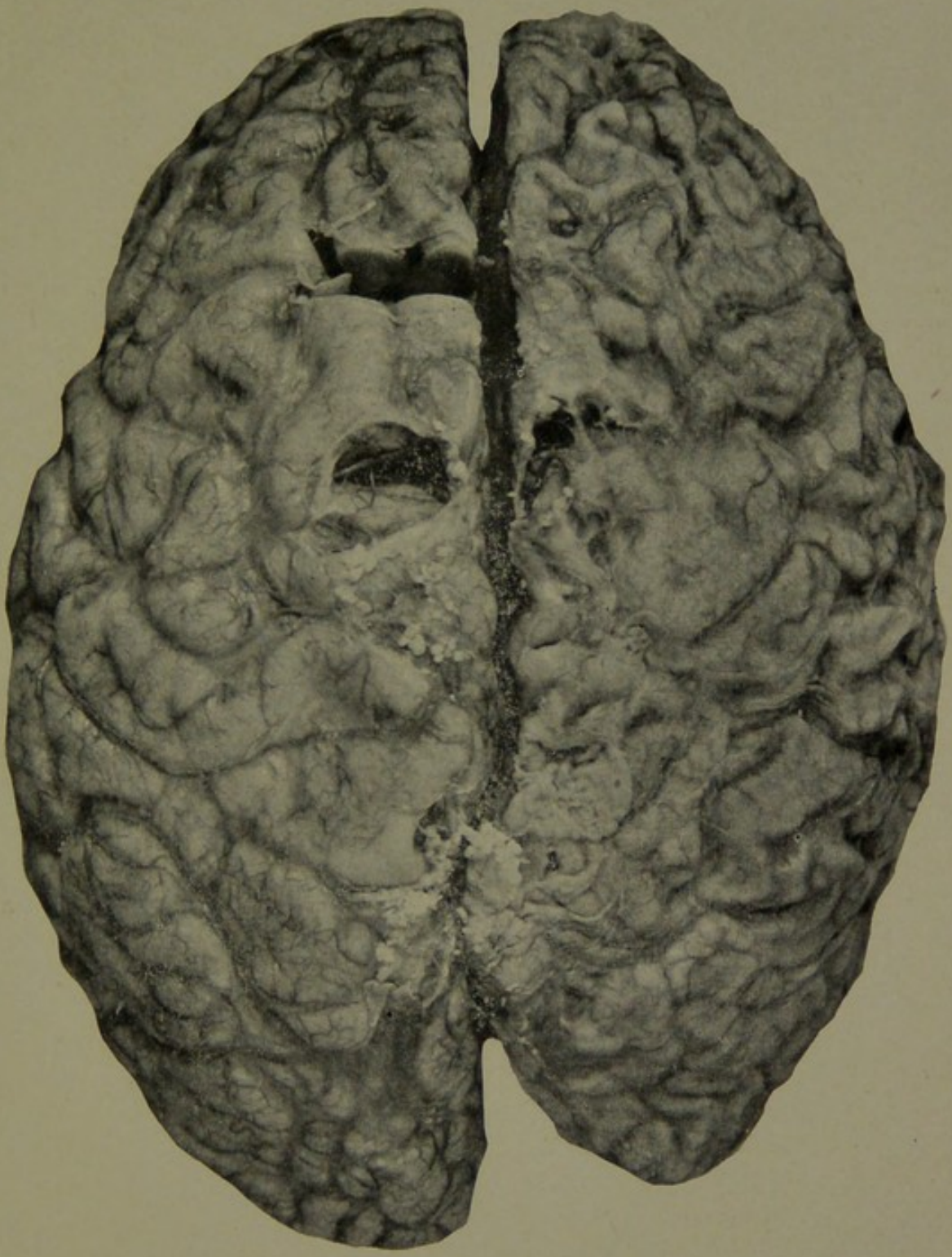


Fig. 6

Case 6 (S. G.) Photograph of the brain showing much more pronounced atrophy of the right than of the left hemisphere.

pyramidal tract and right direct pyramidal tract (Weigert); a section stained with Sudan III showed numerous fat droplets in the left pyramidal tract, none in the right; fibrino-purulent exudate in the pia of the cord.

*Remarks on Case 6.* In this case the focal motor symptoms consisted of weakness and twitching of the whole left side, with terminal contracture of the left hand.

The attacks of the patient were of two kinds: (1) General convulsions of short duration with loss of consciousness not followed by focal symptoms. (2) Twitchings limited to the left side or to part of the left side of short (November 4, 1-½ minute) or long duration, sometimes spreading over a week with irregular intermissions, frequently without loss of consciousness. At other periods there was a mixture of (1) and (2) (June, 1907).

Even after attacks of long duration no gross reduction in the patient's mental condition was noted, but the weakness of the left side was more pronounced (August 29, 1907).

The distribution and nature of the symptoms were not such as could be attributed to disorders in any special vascular area, but appeared to indicate a degenerative and irritative process over an extended cortical area; the various elements in the symptom-picture were of different severity, thus the hemianopia was permanent while the motor symptoms consisted of weakness of variable degree, of twitching of variable distribution, and of terminal contracture of limited extent. This fact would indicate that in the cortex the morbid process was not necessarily always of the same severity over the whole hemisphere.

In view of the severity of the symptoms it was rather striking that this case, in contrast with that of the patient A. S. (Case 8), ran a rather long course; the patient lived for over three years in the hospital, he was for more than four years before death definitely insane, he had begun to show mental symptoms at least nine years before his death. The comparatively striking brightness of the patient even at a late period was no doubt to be explained by the fact that it was not the dominant hemisphere which was so severely affected.

The course of the case consisted of an uninterrupted although slow decline, in contrast with that descent by steps which is frequently attributed to such cases.

The symptoms in this case, therefore, appear to be directly related to the special distribution and severity of the process of general paralysis itself, and not to gross vascular disorders such as those which have been described in the first group of cases. The persistence of the symptoms in this case corresponds with the severity of the pathological changes in the cortex, which were so striking that the right hemisphere showed to the naked eye a much greater reduction than the left hemisphere. In the case A. S. the symptoms were far from showing the same persistence, and on naked eye examination no difference in the hemispheres of the two sides was made out; careful measurements were required to show that the cortex on the suspected side was in certain areas distinctly narrower than that of the corresponding area on the other side. The correlation of the symptoms in the case A. S. with focal severity of the cortical process is strengthened by comparison with the present case S. G. where both the clinical and the anatomo-pathological picture were so much more definite.

The active nature of the changes in the cortex in Case 6 was confirmed by the Sudan III picture of the sections of the cord, showing evidence of recent degeneration in the pyramidal fibers on the left side.

Case 7 (p. 92). R. F., born in 1870, right-handed; alcoholic; syphilis (date?); 1903, inefficient; 1904, speech defect; October, 1904, apoplectiform attack preceded by inability to speak (no details known); April 3, 1905, wandering episode, transitory reduction of speech (no details known); October, 1905, convulsions; June, 1906, transitory weakness of right hand and face; October 23, 1906, without loss of consciousness, attack of weakness of left hand (? leg, ? face), defective reaction to pin-pricks over the whole left side, apparent left-sided hemianopia; the hemianopic symptoms were permanent, the motor and sensory disorder was not demonstrated on October 26, but was present at least in part on later examinations (October 31, November 23, Febru-

ary 7, February 26, March 8); progressive contracture; March 13, 1907, transitory episode with head and eyes turned to the left; April 5, 1907, death.

*Pathological Anatomy.* Diffuse brain atrophy, more marked in the right hemisphere; the right visual area and base of the right temporal lobe posteriorly showed a special degree of atrophy; this condition was not accounted for by any special thickening of the right posterior cerebral artery; the sagittal marrow of the right occipital lobe was somewhat softened; cortex, typical changes of general paralysis, almost more pronounced in the left than in the right paracentral lobule; moderate thickening of the basal vessels; the cord showed no marked degeneration but merely a slight thinning of the pyramidal tracts; endarteritis obliterans of spinal vessels.

*Remarks on Case 7.* In this case we meet with the same problem presented by Case 6 and Case 9, viz., focal symptoms referable to the visual and motor systems without any gross lesion on the basis of a vascular disorder. The right hemisphere appeared to the naked eye somewhat smaller than the left and weighed 440 gr., while the left weighed 472 gr.; the weights were taken after the brain had been in formalin for some time. The thickening of the pia (which was very pronounced) and the cortical atrophy (most marked over the anterior two-thirds of the brain) were more marked over the right than over the left hemisphere. Here, as in the previous case, there is no obvious explanation for the focal symptoms except the cortical process itself with its areas of special severity, the reason for this distribution not being clear. The fact that the severity of the cortical process on both sides was severe, may be taken in conjunction with the clinical observation that during the last two months of his life the patient showed very marked contracture on both sides, lying flexed in an intra-uterine position; the question of contracture is, however, an extremely complicated one. The actual mechanism of the symptoms is not easy to understand, for microscopically the motor area on the one side showed little difference from that on the other: it must be admitted that differences

between the two sides may be present at an earlier stage of the disorder, but be obliterated as the process advances on both sides.

The danger of crude anatomo-clinical correlation may be seen on comparing Case 6 (S. G.) and Case 7 (R. F.); the brain of the former showed a much more pronounced degree of atrophy than that of the latter, although R. F. for several months previous to his death had lived a purely vegetative existence with pronounced contracture, while S. G. was rather bright and responsive until the terminal attack.

The reason for the quick decline of R. F. and the comparatively slow progress of the disorder in S. G., where the brain atrophy finally reached a much more extreme degree, is quite obscure; it is important to remember that in S. G. it was the right hemisphere which showed the most marked atrophy, while the leading hemisphere was much less seriously damaged.

On the whole the course of the disorder in R. F. was one of progressive decline, but after the attack in October his mental level appeared to undergo considerable reduction.

Case 8 (p. 94). A. S., born in 1878, right-handed; alcoholic; syphilis (?1902); 1904, insidious onset of general paralysis; January, 1906, wandering episode, transitory complete inability to talk, leaving a residual aphasic disorder which gradually improved; no hemiplegic symptoms; well marked physical signs of general paralysis; during the summer no progression of symptoms.

October 2, 1906, apoplectiform attack with weakness of the right face and arm, twitching of the right face and platysma; no reaction to pin-pricks over the right arm and leg, apparent right-sided hemianopia, marked reduction of speech with gradual improvement; persistence of twitching and weakness of the right face after disappearance of weakness of the right arm; mental level reduced after the attack.

October 28, 1906, ill-defined attack without loss of consciousness, with very marked reduction of speech and residual peculiarity of intonation, increased weakness of the right face; no special weakness of the limbs; mental level further reduced.

General slight improvement in speech; bed-ridden; progressive deterioration; after January, 1907, unable to speak.

January 16, 1907, twitching of left face and arm with quasi-purposeful movements of the right arm; January 17, twitching of the right arm and first two fingers of right hand, general tremor of left arm, both sides of face twitching; January 21, apparent left-sided hemianopia of short duration (less than two weeks), again reappearing on two occasions (February 26, for less than nine days; March 16 to 25): during the first week in March apparent right-sided hemianopia; during the last two months of life constant contortions of both sides of the face, peculiar high-pitched yelling, no marked difference between the limbs of the two sides; extreme emaciation.

March 25, 1907, death.

*Pathological Anatomy.* No difference between the two hemispheres observed macroscopically; cortex, typical changes of general paralysis; cortical atrophy more pronounced on the left than on the right side (A. C., $F_3$, Calcar.); no focal softening; the basal and cortical vessels were not thickened, except for an occasional spot. The cord, stained by Sudan III, showed a few quite insignificant droplets in the right pyramidal tract, still fewer in the left.

*Remarks on Case 8.* In this case the speech disorder was not merely the usual articulatory disorder of general paralysis but in addition was of the nature of an aphasia. In the episode previous to admission (January, 1906) the patient was unable to speak, and on admission was paraphasic; this condition steadily improved. In the second attack along with right-sided motor symptoms there was inability to talk (except "yes," "no") lasting for over a week, followed by improvement but leaving a considerable residual reduction in spoken speech as compared with the condition of the patient previous to the second attack; there was also agraphia; he understood spoken speech probably up to the limit set by the general disorder.

In the third attack there was increased difficulty in speaking with change of pitch as a residual (cf. *aphasic*

*d'intonation* of Brissaud), and probably further general mental reduction; then insidious progress of the speech difficulty so that for the last month at least the patient did not speak. It was natural therefore to look for some explanation of the above symptoms in that region, the destruction of which is apt to cause motor aphasia; there had been some paraphasia, but the main disorder was of the expressive side of the speech mechanism.

The motor symptoms had throughout been right-sided, either weakness or twitching, but not involving the whole right side.

On October 2 there was weakness of the right arm with twitching of the right face and platysma; for over a week there was weakness or twitching of the right face, platysma, and right arm. The weakness of the face persisted longer than that of the arm.

During the last four months of life, therefore, there were symptoms localized in the right face and arm, and more especially in the face, without permanent paralysis of either face or arm. These symptoms pointed to pathological changes involving the cortical centre for the face and arm or the paths leading from these centres, that for the face being more specially indicated. The coexistence of the special speech disorder with those symptoms seemed to confirm this localization. The sensory symptoms were rather ill-defined and the defects could only be demonstrated by crude methods (reaction to pin-pricks); the defect was liable to be limited to one limb and no frank hemianesthesia was ever present, although on October 2 the patient did not react to pin-pricks over the arm and leg (? partly owing to motor involvement); when he was clearer and again tested, the impairment was limited to the right arm (reaction to pin-pricks).

During the right-sided attack in October the eyes looked to the left and the patient did not wink when feinted at from the right, while he did so when feinted at from the left; this apparent hemianopia persisted for several days. Later (January 21) there was an apparent left-sided hemianopia which persisted for several days and which made

transitory reappearances (reappeared on February 26 and in March); on one occasion there was an apparent transitory right-sided hemianopia. The exact mechanism of this hemianopic disorder was not clear; the patient was not in a condition to give information as to the vision and it was merely deduced from the failure to wink in reaction to stimuli from one side. The limitation of spontaneous movement to one side might be due to an abnormal condition of the frontal cortex of one or the other side and both sides were affected, as was obvious from the twitching. As to the failure to wink in reaction to feinting, the exact weight to be laid upon this symptom is not certain.

In the patient M. D. R. (Case 5) the permanent hemianopia with transitory attacks of complete blindness enabled one to make the correct diagnosis of a disorder in the vascular supply of the occipital lobes, but the hemianopic disorder could be definitely determined by the patient's own statements and was not merely deduced from certain defect symptoms during a condition of general disturbance. In the present case the situation is different; the defect symptoms were much more equivocal and transitory, so that they could not be held to point conclusively to the special involvement of a definite cortical area.

On careful consideration of the convolutions of both hemispheres no difference could be observed macroscopically between the suspected areas on the one side and the corresponding areas on the other (F 3, A. C. Occ.)

Careful measurements, however, of the thickness of the cortex in corresponding areas of the two hemispheres (F 3, Occ. Lobe) were made by Dr. Dunlap and the cortex in these areas was found to be somewhat narrower on the left side than on the right. No explanation was found in the condition of the vessels for the focal symptoms, nor did the nature of the symptoms suggest that they could be explained along the lines of vascular distribution.

The case ran a very rapid course with symptoms pointing specially to active changes in the cortex (twitching) rather than to gross focal destruction with defect symptoms; both sides were obviously implicated but clinically the

symptoms pointed to the greater cortical involvement of the left side, while microscopically the only evidence for this was the slightly more pronounced atrophy.

It is difficult to interpret the attack on October 2, with hemiplegia, hemianesthesia, hemianopia; a complete syndrome of this nature of sudden onset, if permanent, is usually of vascular origin and of capsular localization, the internal capsule and the optic radiation at the same time being involved. Here, however, the internal cupsule and optic radiation showed no special change, and the vessels supplying this area were in satisfactory condition.

Another explanation would be the simultaneous affection of the motor and the visual cortex, determined by an exacerbation of the general paralytic process, possibly extending over the hemisphere although only yielding symptoms from those areas which are not silent, as in the case S. G. (Case 6).

In cases like S. G., where the severity of the cortical process is very pronounced over the whole hemisphere, there may be permanently present the same syndrome, and the evolution of the cortical process manifests itself not so much by apoplectiform attacks as by attacks of twitching which are frequently not accompanied by loss of consciousness, and which may spread over a considerable period of time.

Case 9 (p. 102). J. D., female, born in 1874, right-handed; no history of syphilis; one miscarriage, one stillbirth; July 17, 1908, sudden onset of weakness of left side without loss of consciousness, followed by delirium; slight residual hemiplegia; progressive mental deterioration; January, 1909, delirious episode in hospital; February, 1909, on admission, residuals of left-sided hemiplegia with symptoms of sensory aphasia, general mental reduction; April 15, weakness and twitching of right face, arm and leg, right-sided hemianopia and hemianesthesia; the right-sided symptoms were of short duration; May 20, left-sided hemianopia with reaction to hallucinations in the right field; May 21, general twitching; persistence of left-sided hemianopia; no gross sensory disorder, death on August 23, 1909.

*Pathological Anatomy.* Diffuse brain atrophy, specially

severe in the right occipital lobe, which showed small withered convolutions on the external and under surface of the tip of the lobe, the right calcarine fissure being much shorter than the left; the right posterior cerebral artery was a trifle smaller than the left; no difference between the central convolutions was observed grossly; cortex, typical changes of general paralysis; no focal softening; moderate diffuse thickening of the basal vessels; in the left crus cerebri in the outer third and extending along the periphery of the middle third were extremely numerous fat droplets (Sudan III), the right crus cerebri showed no fat; in the hind-brain there were fewer pyramidal fibers on the right side than on the left; just above the anterior perforated spot external to the optic tract was a smooth-walled lacuna which seemed to separate the peripheral fibers of the tract without injuring them.

*Remarks on Case 9.* In this case, as in the two previous cases, a special degree of atrophy of one cortical area (visual area) had manifested itself in the clinical picture; the slight thickening of the vessel supplying the area was quite inadequate to explain the atrophy. The clinical picture was complex; the left-sided weakness was not accompanied by any gross sensory disorder. The diagnosis of thrombosis in the internal capsule had been made in another hospital; no capsular nor hind-brain lesion was present. Although the anterior central convolution on the right side was not obviously more affected than that on the left, the loss of pyramidal fibers in the right side of the hind-brain indicated the greater affection of the right motor region; it is possible that accurate measurements might have given more direct evidence of this fact. Similarly the most striking evidence of the special involvement of the left temporal lobe, which the symptoms of sensory aphasia had indicated, was derived from the study of the secondary degenerations in the brain stem, where the outer third of the left crus presented a very intense reaction to Sudan III. Thus the clinical picture of symptoms of sensory aphasia in a right-handed woman was explained by the complex pathological picture.

As to the clinical course of the disorder it may be noted that this was characterized by a very rapid and uninterrupted decline, rather than by any special descent by steps.

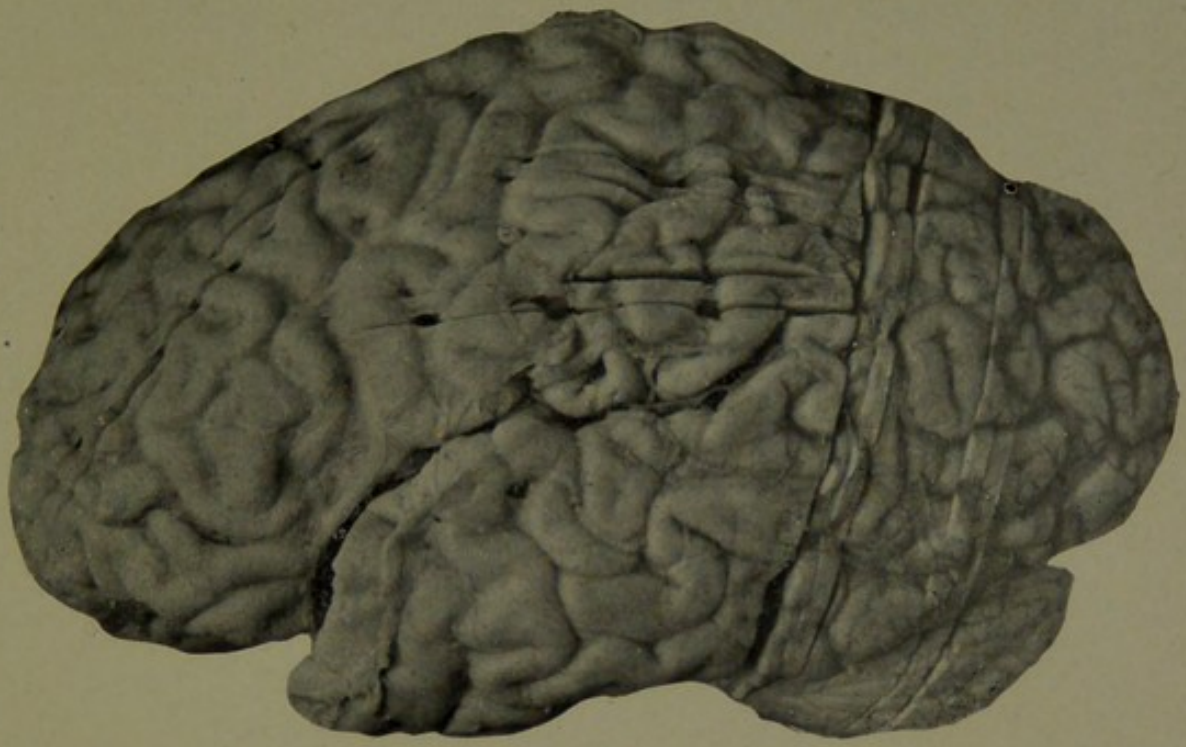
Case 10 (p. 106). G. W., born in 1869, chancre in 1892 (?); Spring, 1905, onset of marked tremor of right hand; February, 1907, fell down steps, was unconscious, lost his speech for several days (diagnosis "cerebral syphilis-general paralysis"); later defective speech, ideas of infidelity, memory defect, headache, diplopia, retention of urine, weakness of the legs; on admission, April, 1908, slight weakness of *right* arm and leg, slight ptosis of the *left* eyelid, slight weakness of the *left* side of the face, marked intention tremor of the right arm, nystagmus; the left pupil reacted less than the right; marked memory defect; ideas of infidelity. Progressive decline, euphoria, loss of depressive ideas, occasional convulsion; terminal convulsions most marked on the right side; death on August 9, 1909.

*Pathological Anatomy.* A generally atrophic brain with special atrophy of the left parietal lobe and the lower third of the posterior central gyrus (vide Fig. 7 A); left temporal convolutions somewhat smaller than the right; cortex, typical changes of general paralysis; in the left parietal lobe the nerve cells had completely disappeared in places; cerebral vessels soft, with only trifling changes.

*Remarks on Case 10.* In this case the clinical symptoms, affection of the left third nerve with weakness and tremor of the right side (Benedikt syndrome), pointed to a lesion in the region of the red nucleus. The cortex was thoroughly studied and presented an extremely typical picture of general paralysis; the localized severity of the process in the parietal lobe could not be explained on the ground of vascular changes.

In his mental condition, pronounced euphoria without megalomanic ideas, the patient resembled very much Case 3 (J. L. W.) and like him had been diagnosed cerebral syphilis previous to admission.

A



Left Right

B

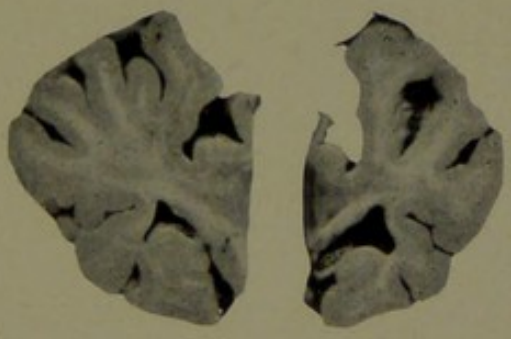


Fig. 7

A) Case 10 (G. W.) Photograph of the left hemisphere, showing pronounced atrophy of the left parietal lobe and first temporal convolution.

B) Case 11 (M. T.) Photograph of corresponding slices from the left and right temporal lobe respectively, showing pronounced diffuse atrophy of the right temporal lobe.

CASE 11 (p. 109). M. T., born in 1868, right-handed; syphilis; December, 1906, cough, frontal headache, hypnagogic visual hallucinations; April, 1907, twitching of left arm for several days, weakness of and feeling of heat in left leg; no residual weakness; Summer, 1907, short attacks of twitching of left hand, with little balls of light dancing in front of his eyes; twice saw objects colored; on admission (June 24, 1907) mild euphoria, memory defect, lack of insight; deep reflexes slightly more active on the left side; no focal attacks in hospital (June to August, 1907); after discharge occasional attacks of twitching of left hand and leg without unconsciousness; little progression of mental symptoms; August 18, 1909, twitching of left arm without loss of consciousness; impairment of sensibility over left hand, shoulder, face; slight residual weakness of left handgrip, left leg slightly stiffer than right; death on November 5, 1909.

*Pathological Anatomy.* Small atrophic brain; the right hemisphere was especially affected, weighed over 40 grammes less than the left; the frontal convolutions, the temporal lobe, and the parietal region were more atrophic on the right side than on the left (vide Fig. 7 B); on the other hand the left anterior central convolution seemed more atrophic than the right, but the right paracentral lobule was more atrophic than the left; cortex, typical changes of general paralysis; basal vessels showed slight diffuse thickening; no focal softening.

*Remarks on Case 11.* In this patient the prominent focal symptoms consisted in transitory affection of the left hand, or of the left hand and leg, the disorder usually consisting in twitching associated with weakness; the right side of the brain was more atrophic than the left. It is impossible to be content with the crude correlation of the right-sided severity of the atrophy with the left-sided symptoms, for the special area indicated by the clinical symptoms, viz.: the right anterior central, was apparently less affected than the corresponding area on the other side. The attacks were of the type which is so familiar in cases of localized severity of the general paralysis process; but, although

cases like S. G. (Case 6) seem to present a simple problem with easy correlation, cases like the present one and like M. H. (Case 14) show how far we are from understanding the mechanism of these focal symptoms.

*Review of Group ii.* The clinical histories and regional distribution of the process in these five cases were sufficiently varied to indicate the difficulty of finding some general explanation for the atypical features of this group of general paralysis. The process was not specially localized in the motor and sensory projection cortical areas in contrast with the usual prefrontal distribution of the process; the areas affected varied a great deal and not obviously along the lines of the special systems of the brain. In some cases specially localized severity of the process was well marked in the usual prefontal area as well as in the unusual area (e. g. M. T. Case 11). The course of the disorder as being a descent by steps rather than a progressive decline has been emphasized by Lissauer and Alzheimer; considerable reduction is apt to follow each attack. Case 8 was the only one of the five cases in which this course was observed; in the other four cases the course consisted essentially of a progressive decline, although in Case 7 considerable reduction followed one attack.

The well known tendency of the same area to be affected in each successive attack was seen in Cases 6, 8, 10; in Cases 7 and 9 this tendency was not so striking. Case 8 did not present such clean-cut permanent focal symptoms after the attacks as the other four cases.

In two cases (Cases 7, 8) the insidious onset of general deterioration had been noted before the occurrence of the focal attacks.

In this group as in the first group there occurred a variety of attacks, apoplectiform, epileptiform, and epileptiform with Jacksonian features.

Apoplectiform attacks occurred as the earliest symptom in Cases 7 (R. F.), 9 (J. D.), 10 (G. W.). In the first attack of Case 7 the diagnosis of *epilepsy* had been made on his admission to a general hospital, and of *acute alcoholism* on his discharge. In the first attack of Case 9 the diagnosis of *thrombosis of the internal capsule* had been made;

no focus of softening was found on anatomical examination. In the first attack of Case 10 the patient was diagnosed "*Cerebral syphilis-general paralysis*". These facts are sufficient evidence of the difficulties presented by these cases in the early stages.

In Case 6 (S. G.) the history was not sufficiently definite to warrant a statement as to the nature of the early episode, in which there was weakness of the left leg.

In only three of the six cases were general epileptiform convulsions observed (Case 6, S. G.; Case 7, A. S.; Case 10, G. W.).

In five of the cases (Cases 6, 8, 9, 10, 11) there were attacks of which a prominent feature was localized twitching, extending over a greater or less period, with or without loss of consciousness, in relation to or independent of general convulsions, leaving (e. g. Case 6, S. G.) or not leaving (e. g. Case 8, A. S.) definite residual weakness.

It may be noted that the patient (Case 7, R. F.) who did not show any indication of this localized twitching presented the most extreme degree of contracture of all the patients, and died in the intra-uterine position.

In all the five patients, who presented this localized twitching, the tendency of the twitching to affect the same regions in each succeeding attack was very striking; this feature was less marked in Case 9 than in Cases 6, 8, 10.

The pathological anatomy of this group presents numerous interesting problems, but a discussion of the latter would involve too many hypothetical considerations to come within the scope of this communication.

GROUP III. *General paralysis with focal symptoms associated with trauma.*

Case 12. F. S.
Case 13. P. D.

CASE 12 (p. 112). F. S., born in 1855; gonorrhea admitted, syphilis denied; 1875 (approx.), severe trauma, transitory hemiplegia, permanent anosmia with impairment of taste and hearing; change of disposition; 1896 (approx.), epileptiform convulsions; 1896 to 1908, occasional epilepti-

form convulsions with numerous minor attacks; 1906, indifferent towards loss of work, mild mental deterioration, impairment of memory; on admission (August, 1909), mild mental reduction with considerable insight, no expansiveness; anosmia, impairment of sense of taste, defective hearing on the right side; knee-jerks active, speech and writing defective, tremor, lymphocytosis and positive Wassermann reaction of the cerebro-spinal fluid; September 14, 1909, transitory attack of confusion; progressive decline; November 15, 1910, apoplectiform attack, sign of Babinski on both sides; November 19, 1910, death.

*Pathological Anatomy.* The right cerebral hemisphere appeared slightly smaller than the left; an old traumatic lesion had involved the olfactory surface of both frontal lobes (vide Fig. 8) so that neither olfactory lobe and only the posterior half of the left olfactory tract could be found; there was a gutter-like lesion on R. $T._3$ about 4 cm. long, and a smaller lesion on R. $T._2$; cortex, typical changes of general paralysis; no focal softening; slight thickening of the cerebral vessels.

*Remarks on Case 12.* The point of special clinical interest in this case was the date of onset of the convulsions; the first convulsion occurred twenty years after the trauma, and ten years before the insidious onset of general paralysis. The interpretation of this symptom, therefore, was difficult; was it to be considered as a late result of the trauma, which had earlier caused the development of an epileptic disposition, or was it to be considered as the earliest expression of the cortical changes of general paralysis? The exact rôle played by the two factors could not be estimated.

The early diagnosis of the case was difficult but as the case progressed the clinical picture was quite classical.

Case 13 (p. 114). P. D., right-handed, born in 1859; no history of syphilis; February, 1905, after severe trauma unconscious for four days; no focal symptoms; post-traumatic irritability and headache; early in 1906 forgetful, unable to recall names, "peculiar in his talk"; March 21, 1906, epileptiform convulsions with right-sided hemiplegia; residual paraphasia without weakness of the right side. May,

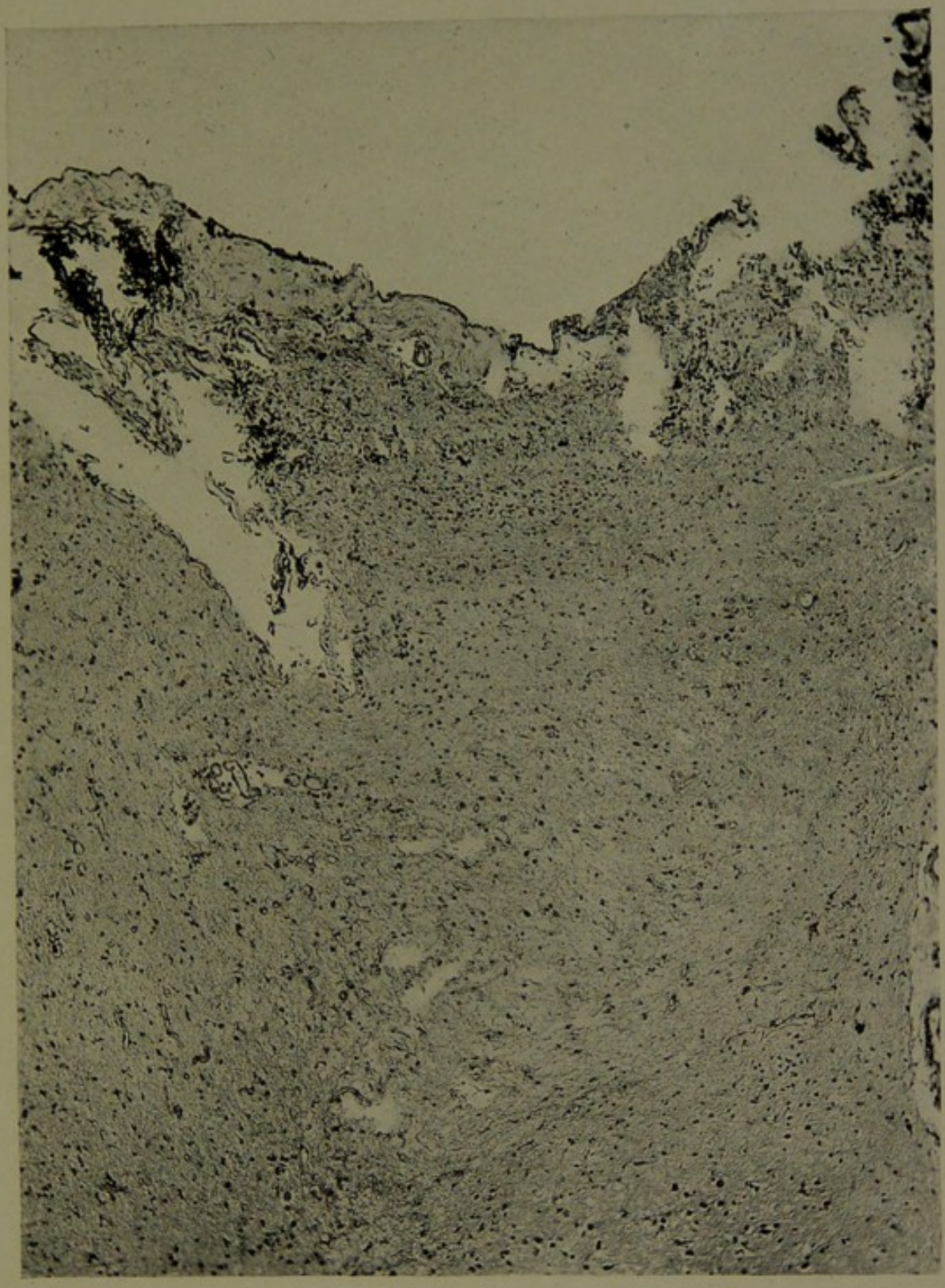


Fig. 8

Case 12 (F. S.) Microphotograph of the left gyrus rectus, with old traumatic lesion. The tissue shows a somewhat loose reticulum, sparse neuroglia nuclei, disappearance of nerve-cells. The surface is somewhat ragged owing to the difficulty of removing the brain without tearing.

Compare this old lesion with that of Case 13 (P. D.) shown in Fig. 10, 11.

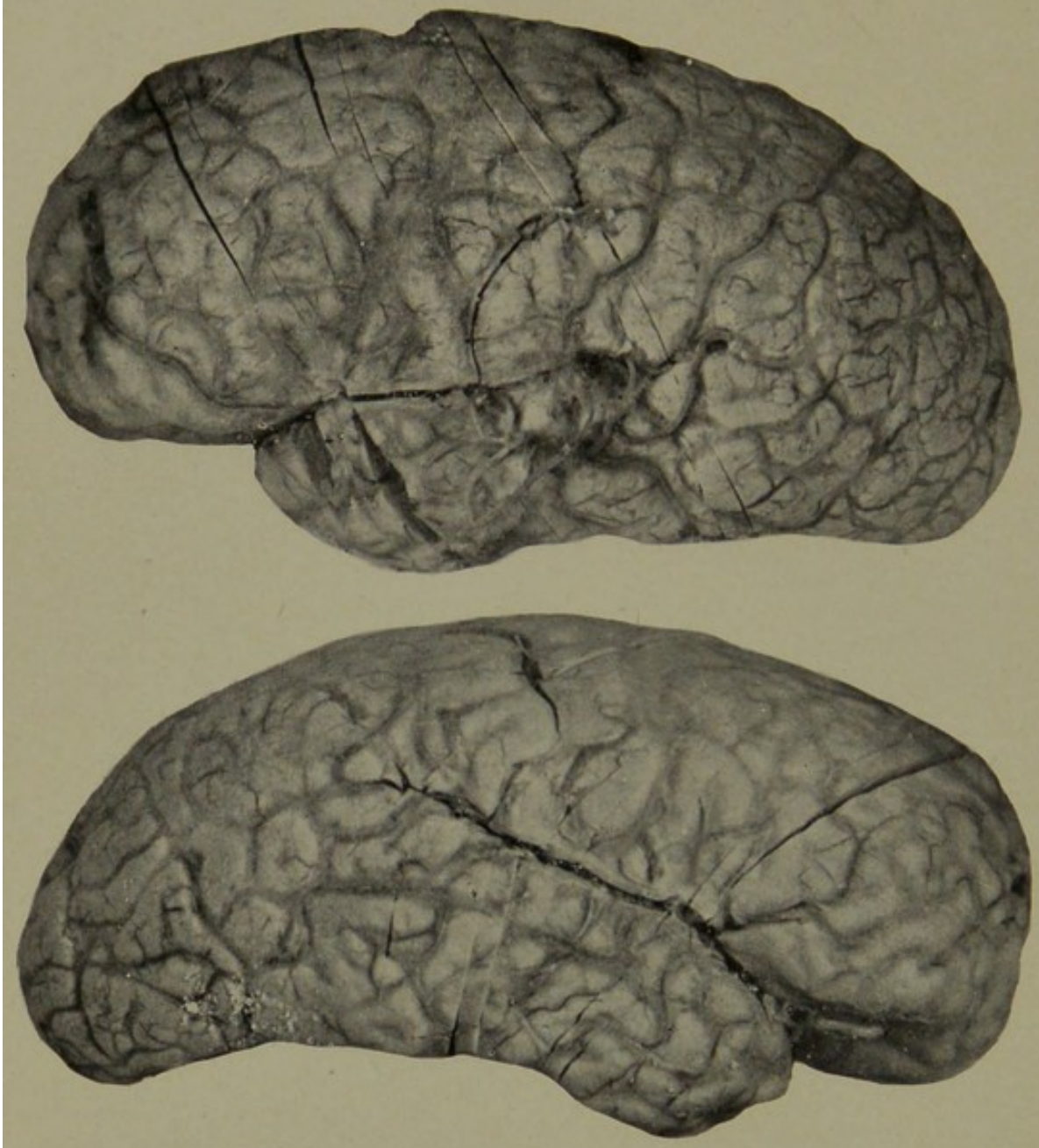


Fig. 9

Case 13 (P. D.) Photographs of the right and left hemispheres, showing the cortical softening in the left temporal region; the cortex here was rusty, depressed, soft.

1906, fracture of the base of skull, delirium; euphoric dementia with sensory aphasia; no weakness of face nor limbs, no gross sensory defect, K. J. absent, A. R. pupils. July 10, right-sided convulsions with transitory inability to talk, weakness and impaired sensibility of right face and arm, with spasticity of the arm. October, aphasic symptoms still more pronounced, weakness of right side of face, difficulty of innervating right hand; extremely limited utterances with an occasional appropriate phrase. After November, no utterances except "yes" and an oath; contracture of fingers of right hand without weakness or gross disorder of pain sense; later convulsions which specially affected the right side; in Spring progressive contracture of right leg; May 3, 1907, death.

*Pathological Anatomy.* Medium-sized, somewhat atrophic brain, with the atrophy and pial thickening more marked over the anterior two-thirds; a brownish area of cortical softening involved the anterior half of the left temporal lobe exclusive of the temporal tip (Fig. 9); $T_1$, $T_2$, $T_3$, were all affected; the lesion consisted of a superficial softening and extremely slight depression without break in the pia; transverse temporal convolutions a little wrinkled, but firm and not discolored; slight diffuse thickening of the basal vessels; no focal softening apart from the above lesion.

*Remarks on Case 13.* In this case neither the clinical nor the anatomical picture is so easy to interpret as in the preceding case. The patient had a severe trauma, followed by unconsciousness, several months before the onset of the mental symptoms, and of a certain peculiarity in his talk. It is extremely improbable that the later focal symptoms had any relation to this first trauma, for in that case one would expect the maximum of focal disturbance to have occurred immediately after the trauma, with gradual restitution partial or complete; in this case the sequence of events was different, for the first traumatic episode was characterized by no focal symptoms. One year later, two months before the fracture of the base of the skull, the patient had a general convulsion with residual paraphasia.

It seems probable that this attack, with its residual symptoms, arose on the same basis as similar episodes in general paralysis with localized severity of the paralytic process.

Two months later the fracture of the skull led to further interference with the speech mechanism, and this disorder became progressively worse until spoken speech was reduced to a minimum. The sequence of events concerning the left motor area consisted in early attacks of weakness with later progressive contracture, the face and hand being more consistently affected than the leg.

The histological picture was somewhat difficult to interpret; the softened area (Fig. 10, 11) did not correspond in its structure to the softened specially atrophic convolutions of case 10 (G. W.) where the nerve cells had dropped out altogether in places, but where there had been a massive proliferation of the neuroglia (vid. Fig. 12 from another case). In the present case the softened area presented in places an open mesh-work, with only moderate proliferation of the neuroglia in the neighborhood. No gitter-cells were demonstrated, and this fact would be somewhat unusual in a case of gross destruction of tissue of such recent duration (assuming that the fracture in May, 1906, was the cause of the lesion).

It seems probable, therefore, that the temporal area was especially affected before the trauma in May, 1906, and that, as a locus minoris resistentiae, on the occurrence of the trauma it suffered a further reduction. The histological picture, however, does not give much evidence of the actual paralytic process being specially severe in this region.

The accompanying photographs illustrate the difference between the lesion in this case and that of Case 12; in the latter, however, the lesion had occurred 35 years before death.

*Review of Group iii.* Little need be said of this group in addition to the remarks on the individual cases. In Case 12 we have to deal with a brain, with unequivocal evidence of an old traumatic lesion, presenting the typical histopathological changes of general paralysis. That the trauma may have had an influence in rendering the brain more susceptible to the causes of general paralysis can not

Fig. 10

Case 13 (P. D.) Microphotograph of L. $T._1$ showing pronounced cortical atrophy with disappearance of the nerve-cells; the dark strands in the cortex and sub-cortex represent neuroglia masses, frequently in association with vessels.

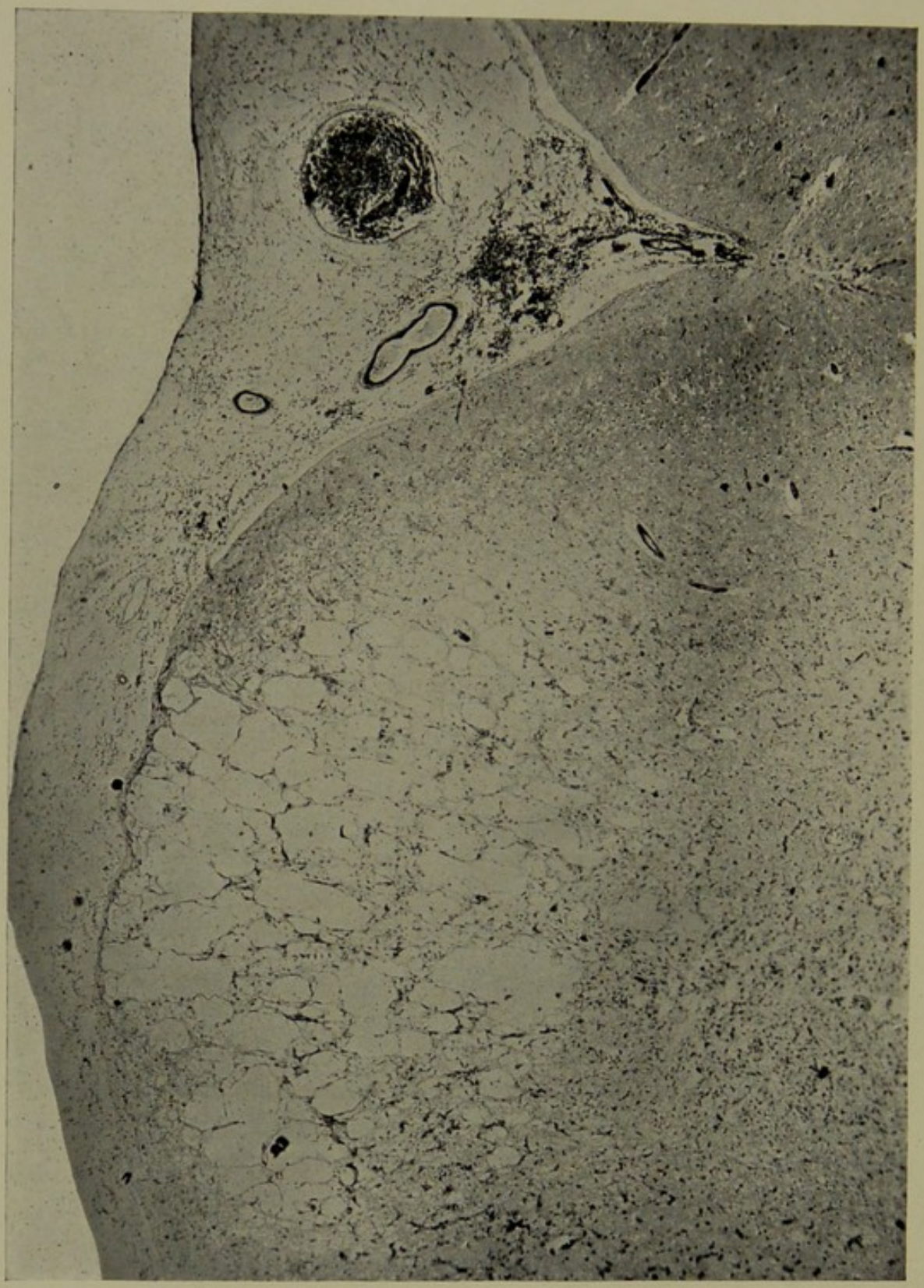


Fig. 11

Case 13 (P. D.) Microphotograph of the lesion in L. $T._1$, showing still more pronounced destruction of the cortex. The tissue is composed of an extremely loose glia reticulum with complete disappearance of nerve-cells.

be denied; the post-traumatic change in disposition and the onset of convulsions at least 10 years before the insidious beginning of the mental deterioration were evidence of the great importance of the trauma. The general paralysis, however, presented a classical picture in which there was nothing to indicate an unusual origin; the neurological symptoms due to the trauma did not essentially modify the general paralysis. Similarly in the brain the lesions of the one process existed side by side with those of the other, without the nature of the general pathological changes having been specially modified. Such a case, therefore, in no way justifies the use of the term "traumatic general paralysis", which unfortunatety is occasionally seen in the literature.

In the second case the exact nature of the focal lesion has been described and the probable relation to the trauma discussed. It was probable that the severity of the destruction in this area which expressed itself in the clinical picture, was to be explained by the trauma. The trauma had modified the symptomatology, and possibly hastened the course of the general paralysis.

Even in cases like this the use of the careless term "traumatic general paralysis" has to be deprecated; we have to deal with a disorder presenting a characteristic symptomatology and pathological anatomy, and the fact that complicating factors may modify both the clinical and pathological picture does not alter the conception of the disease nor warrant us in giving to these factors the etiological importance implied in the term "traumatic general paralysis"; one might with equal cause talk of an "alcoholic tuberculosis". That trauma may precipitate the onset of general paralysis and hasten its course is recognized; legal decisions in Germany have awarded compensation on this ground, although it was not maintained that the disease in itself was of traumatic origin (Gerlach[5]).

The pathological findings in Case 13 would support this point of view, for while there was no doubt as to the nature of the fundamental disorder the influence of the trauma could be also clearly demonstrated.

GROUP IV. *General paralysis with focal symptoms without adequate explanation, such as vascular lesions or localized severity of the cortical disorder.*

Case 14. M. H.
Case 15. W. S.
Case 16. M. L.
Case 17. A. H.
Case 18. N. F. T.
Case 19. W. B.

CASE 14 (p. 118). M. H., female, right-handed, born in 1857; no history of syphilis; 1902-3, slight change in disposition; July 10, 1903, transitory cramp and stiffness of right hand and leg (? face); August 31, transitory cramp of right arm and hand, followed by delirium; on admission September 5, 1903, mild euphoric dementia, slight weakness of the right hand grip, right triceps reflex a little more active than the left, no weakness of face nor leg observed, no gross sensory disorder, defective speech and writing; defective pupillary reaction to light; lymphocytosis of the cerebro-spinal fluid (October 25, 1903).

September 27, attack of weakness of right lower face and arm, without weakness of leg, sense of pain impaired over the right arm, deep reflexes more active on the right side than on the left; eyes turned to the right, no winking on feinting from the right, nystagmus; inability to speak, no evidence of understanding spoken commands; during the following week steady return to previous level through a period of jargonaphasia and paraphasia with perseveration, with peculiar movements of the right hand; April 22, 1904, attack of weakness of the right hand, delirium; on readmission April 27, 1904, right face slightly flattened, no weakness of arm nor leg, right triceps reflex more active than the left; May 9, two attacks, the first characterized by pallor, inability to talk, impaired sensibility on the right side, the second by purposeless movements of the right arm, and of the head and eyes, with impaired sensibility over the right side, nystagmus, and residual paraphasia; after June, attacks of dulness with apparent helplessness of the right hand, progressive deterioration; June 27, transitory want of

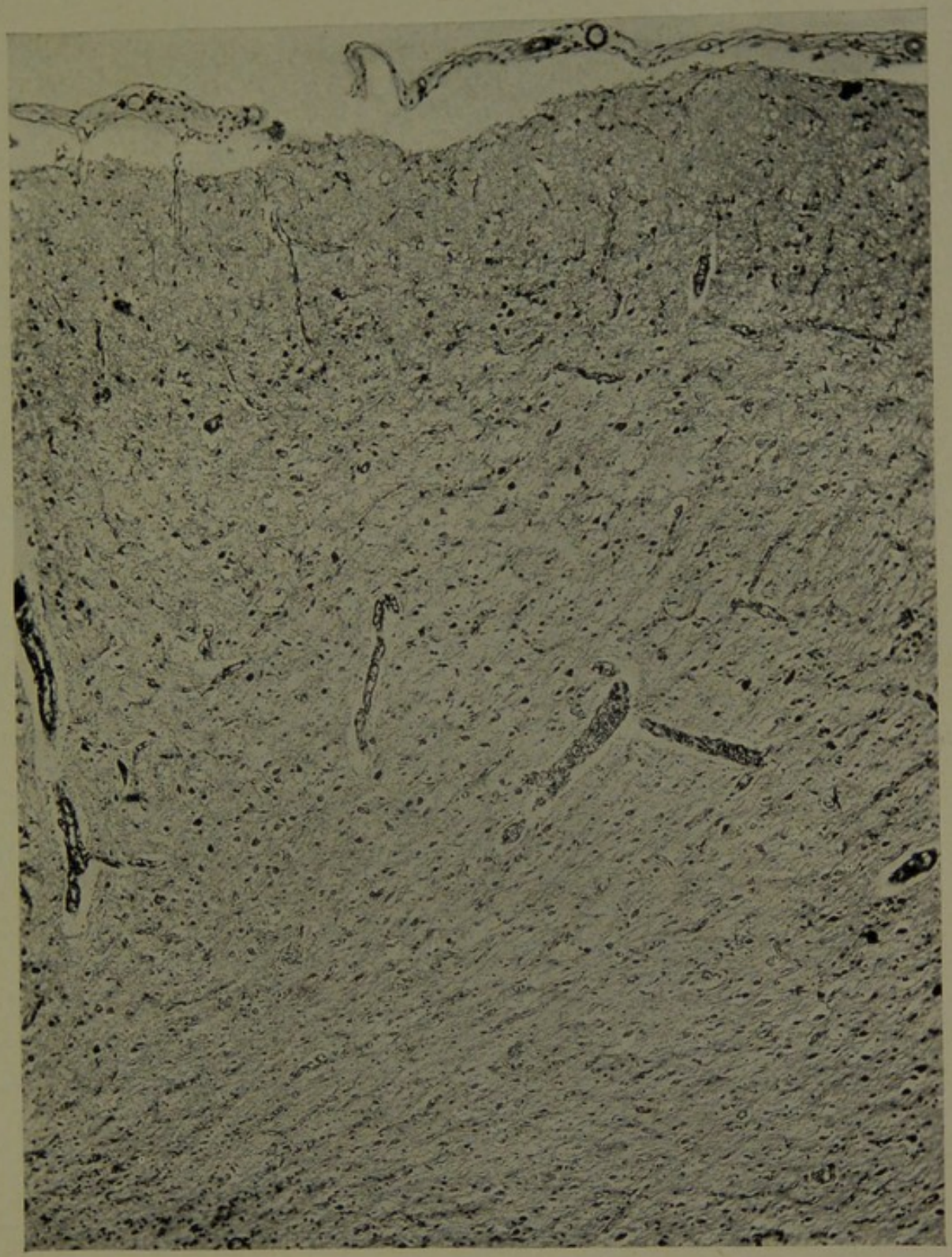


Fig. 12

Microphotograph of cortex in general paralysis, showing a very severe and advanced stage of disorder: to be compared with vascular lesion (Fig. 5); traumatic lesion (Fig. 8); and atypical traumatic lesion (Fig. 11).

utilization of right arm; June 28, two attacks of twitching especially involving the right face and arm, without increased residual weakness; August 5, transitory attack of dulness with weakness of the right arm; October 9, attack of dulness with accentuation of the speech defect, and reference to her right hand; December 16, after period of drowsiness and reduced talkativeness, transitory anarthria, inability to use right hand, with residual paraphasia and preseveration which persisted; during 1905 further deterioration without change in the neurological status, slight flattening of the right face, no gross weakness of the right arm, right elbow-jerk stronger than the left, right hand usually held on chest, slight scraping of right foot on walking; no further attacks observed. Death on April 3, 1906.

*Pathological Anatomy.* General cortical atrophy, most marked in the prefrontal region; to the naked eye the left central convolutions appeared slightly more atrophic than the right; cortex, typical changes of general paralysis; on large sections taken through the arm area and F 2 the histopatholigical changes on the left side were not more pronounced than on the right side; basal vessels thin, unusually small; some subcortical vessels showed narrowing of lumina with colloidal changes.

*Remarks on Case 14.* In this case the symptoms were of exquisitely focal nature during the long period of three years, but, although the patient had extremely numerous attacks localized in the right hand, or right hand and face, with involvement of the speech mechanism, there was no permanent gross reduction of the strength of the right arm even during the terminal stage of the disorder. In this respect the case may be compared with the previous case (P. D.) where similarly, even in the most advanced stage of the disorder, no weakness of the right hand was demonstrable, while contracture was progressive and severe. In both cases there was a defect of utilization of the limb which was not due to a crude loss of motor power.

A case where focal symptoms have been so pure seems to be specially promising for purposes of histopathological

research; the attacks, however, though frequent and exquisitely localized, left such trifling permanent focal residuals that the negative results of the microscopical examination are not surprising. In the light of such a case we must try to think in less crude terms of the mechanism of the symptoms; it is difficult, however, to give any precision to our conception of these episodes.

Very thorough topographical studies of the cortex in this case were undertaken by Dr. Dunlap; he found no structural differences which might account for the onesided symptoms; the two hemispheres showed apparently equal involvement (except that the left central convolutions appeared to the naked eye more atrophic than the right); even in the relative count of neuroglia nuclei and in the number of Betz cells no difference between the two sides was observed. The large section on the left side was, however, taken from a level towards the upper limit of the region indicated by the clinical symptoms.

CASE 15 (p. 124). W. S., born in 1855, right-handed; very alcoholic; ? syphilis; 1904, defective speech, peculiar behavior, onset of frequent attacks of inability to talk, with weakness of the right hand; 1905, pronounced mental dilapidation, classical general paralysis; December 17, 1905, attack of weakness (no detailed examination), paraphasia for several days; January, 1906, speech extremely defective; February, transitory general improvement; death September 18, 1906.

*Pathological Anatomy.* Cortical atrophy, most pronounced in prefrontal region; cortex, typical changes of general paralysis slightly more marked in the left hemisphere than in the right; no focal softening; slight diffuse thickening of the basal vessels which were small.

*Remarks on Case 15.* The only attack with focal symptoms observed in the hospital consisted of a paraphasic period of a few days duration; numerous transitory attacks with weakness of the right hand and inability to speak were said to have occurred previous to admission. No focal softening on the basis of vascular disorder was present, no special localized atrophy of the brain was observed, and

the slight difference in the microscopical sections taken from the two hemispheres could not be used as an adequate explanation of the clinical symptoms.

Case 16 (p. 126). M. L., born in 1872, right-handed; alcoholic; syphilis of unknown date; 1900, delirious episode after drinking; 1904, insidious onset of queer behavior at work; September, 1904, sudden dysarthria, followed by improvement; winter, 1904–05, deterioration of conduct; March, 1905, sudden dragging of right leg, awkwardness of right hand, paraphasia of one hour's duration; December 17, transitory weakness of the right arm and leg; January 9, 1906, transitory numbness of legs and of left wrist; January 14, 1906, transitory weakness of left hand; January 25, transitory stiffness of the left arm; March 20, transitory left-sided weakness of ten minutes duration; March 21, transitory numbness and weakness of right hand; November 17, transitory weakness of right face, arm, leg; December 3, episode of weakness; progressive enfeeblement; April 15, 1907, death.

*Pathological Anatomy.* Moderate degree of convolutional atrophy; no focal softenings; cortex, typical changes of general paralysis; no localized severity of the cortical changes demonstrated, but there was a striking neuroglia reaction in the white matter of some convolutions; cerebral vessels in good condition.

*Remarks on Case 16.* In this case numerous details of histopathological interest were observed, but no localized changes which one could correlate with the clinical symptoms; in view of the transitory nature and variable distribution of the symptoms this was to be expected.

Case 17 (p. 130). A. H., born in 1885; alcoholic; syphilis (at 28?); 1905 precordial pain, sleepless, restless, unable to work; progressive deterioration; on admission well-marked signs of general paralysis, knee-jerks absent. April 1, 1907, attack of left-sided weakness without loss of consciousness; apparent left-sided hemianopia; ?sensibility. April 4, transitory ataxia of right arm; persistence of left-sided weakness until death, April 14, 1907.

*Pathological Anatomy.* Slight convolutional atrophy, no gross evidence of localized special severity of the process; cortex, typical changes of general paralysis; no focal softening; cerebral vessels in good condition.

*Remarks on Case 17.* In this case the focal symptoms were of extremely short duration, persisting during the last two weeks of life; no vascular disorder was present to account for the symptoms; in view of the brief duration of the symptoms it was not expected that there would be anatomical evidence of severe cortical changes.

CASE 18 (p. 131). N. F. T., born in 1860; alcoholic; no history of syphilis; delirium tremens in 1900; onset of mental symptoms unascertained; workhouse in 1904; January, 1905, on admission classical general paralysis; September 1, 1906, left-sided convulsions followed by left-sided weakness and impaired sensibility; the weakness was of several hours' duration, the sensory disorder lasted more than a day; September 18, sudden weakness of right side, inability to speak; no residual after a few days; October 4, weakness of right hand (?face, leg), lasting one week, tremor and twitching of extremities; decubitus; November 9, 1906, death.

*Pathological Anatomy.* Acute meningitis, grafted on a chronic meningo-encephalitis, secondary to decubitus and purulent bronchitis; cortex, typical changes of general paralysis; no focal softening, no gross evidence of localized severity of the cortical process; basal vessels slightly atheromatous.

*Remarks on Case 18.* The case presented considerable interest from the histopathological point of view; the acute infective process remained confined to the meninges, and interfered comparatively little with the layer of the pia next the cortex, which presented the exudate characteristic of general paralysis.

The absence of gross focal changes and of evidence of localized severity of the cortical process of general paralysis is not surprising in view of the transitory and inconstant nature of the clinical symptoms.

Case 19 (p. 133). W. B., born in 1860, congenitally left-handed; temperate; syphilis (? date); August, 1904, episode of forgetfulness and difficulty in writing; onset of classical general paralysis; November 22, 1905, sudden onset of weakness of left arm (face? leg?) with paraphasia and inability to write his name; later, doubtful hemianopia, inability to recognize objects with the left hand (? sensibility), difficulty in carrying out definite movements with the left hand, peculiar attitudes of left hand; duration of left-sided symptoms less than two weeks; July 17, 1906, general convulsions with residual weakness of left face, arm and leg, apparent left-sided hemianopia, speech difficulty; coma; death on July 19, 1906.

*Pathological Anatomy.* No gross lesion of the brain; well marked atrophy of the convolutions especially in the frontal region; cortex, typical changes of general paralysis, the perivascular infiltrate being as well marked in the central as in the frontal convolutions; cerebral vessels were thin, without evidence of atheroma; in the cord both crossed pyramidal tracts showed considerable fatty degeneration (Sudan iii) indicating progressive myelin decay; there was little difference on the two sides; the posterior columns showed the lesions of early tabes.

*Remarks on Case 19.* This case ran an unusually rapid course and death occurred after a series of convulsions, with special involvement of the left side; in the first attack a left-sided syndrome was also present—weakness and astereognosis of the left hand, doubtful hemianopia, paraphasia. The paraphasia formed part of the left-sided syndrome, as the patient was congenitally left-handed, and only right-handed by education.

The cortical changes in the motor cortex were as pronounced as in the frontal region, which is unusual; the infiltrate appeared to be about equally prominent in the left and right paracentral lobule. In a Weigert preparation both the crossed pyramidal tracts showed a very marked loss of nerve-fibres, but it was difficult to say which side was the more affected; in a Sudan iii preparation both the crossed pyramidal tracts showed considerable fatty degen-

eration, indicating a progressive process of myelin decay; there was little difference on the two sides. There was thus no microscopical evidence that the focal symptoms were in relation to a specially intense destructive process in the right motor cortex.

On the other hand the cerebral vessels were in excellent condition and no focus of softening was present. The mechanism of the attacks, therefore, in this case was quite obscure.

*Review of Group iv.* The study of the previous cases has already shown that even where the symptomatology promised the possibility of clinico-anatomical correlation, the latter was frequently possible only in a rather restricted sense; in Case 8 (A. S.), where the symptoms pointed to the special involvement of a certain area in the left hemisphere only microscopical measurements detected a difference between that area and the corresponding area on the right hemisphere; in Case 9 (G. D.) recourse was had to the study of the secondary degenerations in order to obtain even a crude correlation.

If the anatomical study frequently yield so little to explain the atypical features of the symptomatology in cases where the attacks have been numerous, severe, consistent in the localization and even with permanent residuals, it is not to be wondered at that, in cases where the attacks are either fewer, less severe, more variable in their localization or without permanent residuals, the study of the pathological anatomy leaves many questions unanswered.

Perhaps the most striking case in this group and in one way the most disappointing one, is Case 14 (M. H.) where, notwithstanding numerous exquisitely focal attacks extending over a period of almost three years, the microscopical examination furnished no clue to the special symptomatology; it is true that the absence of any gross focal residual was one point which made the possibility of definite correlation somewhat problematical.

No group of cases is better suited to emphasize the limits of our present knowledge of general paralysis. It warns us against making too much of the anatomo-clinical corre-

lation in cases where circumscribed lesions are found; we must confess that, even in presence of a circumscribed lesion, our conception of the actual mechanism of the focal symptoms is often a hypothetical construction on the basis of rather inadequate facts.

## Summary.

The introduction of the cytological and serological examination of the cerebro-spinal fluid into psychiatric procedure, and the establishment of a satisfactory histopathological criterion of general paralysis have within the last ten years necessitated a much more critical attitude and the adoption of a more adequate standpoint. The histopathological criterion, introduced by Nissl and Alzheimer, may not be final, but its provisional adoption is necessary at present if there is to be a common understanding in the discussion of general paralysis.

In the discussion of the main issues cases of general paralysis reported before 1900 should be used with the greatest reserve, even although the statement is made that the diagnosis was confirmed by post-mortem examination. One must accept with reserve purely clinical observations even if recent, especially if they do not include the examination of the cerebro-spinal fluid; to publish as cases of general paralysis cases of atypical course, whether influenced by therapeutic measures or not, without an adequate examination of the cerebro-spinal fluid, indicates a want of realization of the difficulties of differential diagnosis.

It is sometimes impossible to make a clinical diagnosis between general paralysis and cerebral syphilis; the question, whether in some rare cases the anatomical diagnosis too is impossible, is outside of the scope of this communication; the answer to the question does not affect the value of the histopathological picture as furnishing the only safe provisional criterion, which makes possible a common understanding.

The difficulty of differentiating between general paralysis and cerebral syphilis is insufficiently realized, even by

serious authors whose opinions carry great weight; thus it is probable that many cases are wrongly used in formulating conclusions as to the symptomatology, the course, the serological reactions of general paralysis.

Among the cases of general paralysis presenting atypical features and considerable diagnostic difficulties is the large group of cases of general paralysis with focal symptoms; these cases deserve study, not only from the point of view of differential diagnosis, but also for the light which they throw on the evolution of the disease.

The focal symptoms may be more or less irrelevant to the process of general paralysis, e. g., in general paralysis with traumatic lesions. Trauma may precede the development of the general paralysis and perhaps act as a predisposing cause; on the other hand the trauma may act on a brain already disorganized by general paralysis. In neither case is the term "traumatic general paralysis" justifiable.

The focal symptoms may be more or less irrelevant to the general paralysis but may be due to lesions secondary to vascular changes, frequently of syphilitic origin. These lesions may develop many years before the onset of the general paralysis; they may develop within the prodromal period of the general paralysis; they may develop when the general paralysis is already well established. The consideration of such cases is important in trying to formulate a conception of the evolution of the disorder. Where the development of the vascular lesion accompanies the evolution of the general paralysis, there are two processes side by side and this is one additional fact in favor of the suggestion that general paralysis may not be a unitary process, but that part of the process may be merely a late syphilitic manifestation. This observation must be remembered in connection with certain claims for the benefit of anti-syphilitic treatment in general paralysis.

Vessel changes may cause symptoms not only through their effect on the nutrition, but directly through the pressure of dilated and tortuous vessels on the nervous tissue.

The symptomatology of general paralysis with focal lesions on the basis of vascular changes is frequently not

modified by these changes except through the occurrence of apoplectiform attacks and the presence of residual symptoms.

In a third large group focal symptoms develop in the absence of gross vascular changes, and appear to be in relation to the specially localized severity of the process of general paralysis in certain cortical areas (Lissauer's Atypical General Paralysis). The factors which determine this atypical distribution of the morbid process in the cases reported above were quite obscure; there seemed to be not the slightest tendency towards a systemic distribution. The correlation of the anatomical changes with the symptomatology is frequently rather crude. In attempting to correlate the clinical symptoms with the anatomical findings one is warned by the divergence of the cases to be extremely conservative.

The course of the disorder in the majority of the cases here reported consisted of a progressive decline and was not characterized by that discontinuous reduction, each step of which follows upon an attack, which is sometimes observed in similar cases. Specially characteristic of this group of general paralysis are the epileptiform convulsions of Jacksonian type.

In a fourth group are included cases in which no correlation of focal symptoms with anatomical findings could be made, and which give a further warning against too dogmatic correlation in cases where some lesion happens to be found. We have as yet no adequate data on which to form any valid conception of the mechanism of these attacks.

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# APPENDIX.

## CLINICAL OBSERVATIONS.

### List of Cases.

*Group i.* General paralysis with focal symptoms on the basis of vascular lesions:



*Group ii.* General paralysis with focal symptoms on the basis of localized severity of the general paralysis process:



*Group iii.* General paralysis with focal symptoms associated with trauma:



*Group iv.* General paralysis with focal symptoms without adequate explanation, such as vascular lesions or localized severity of the cortical disorder:



Case 1. W. M., 42, night watchman; admitted to Manhattan State Hospital, December 9, 1903.

*Family History.* A paternal cousin died in an insane asylum.

*Personal History* The patient, a right-handed man, was born in Ireland in 1861; after leaving school he worked in hotels, and probably drank freely. At 25 he married a woman of doubtful reputation, who became insane after three years and died in a sanitarium six years after marriage; he indulged in excessive sexual intercourse. No history of syphilitic infection was obtained; as far as known his wife was never pregnant. On the death of his first wife the patient immediately remarried; his second wife was never pregnant.

At the age of 33, in August, 1894, while in a car, his *left* hand "fell asleep;" he commenced to pat it; he then found that his leg was "asleep." As he left the car he stumbled and was considered drunk, but said "I wish I was, it is something worse." He was driven home, but was able to walk with assistance to the house; the hand seemed worse than the leg; he had no trouble with his speech, and was able to swallow quite well. After a short time in bed (duration uncertain) he appeared to be well; his wife noticed no residual weakness of the left leg; so far as she remembered the left arm showed no weakness but occasionally a little tremor when he was excited. Some residual weakness was observed, however, when the patient was treated for osteitis of the right tibia (due to old trauma) in the New York Hospital in February, 1895; "the patient has *almost* recovered control over hand and leg."

In 1902 the patient became restless, sleepless and nervous, and complained of pain in his temple; in Spring, 1903, he became lazy, shirked his work as night watchman, was content to be supported by his wife. In September he began to show absurd behavior; he dressed himself at night, accused his wife of infidelity, chased her with scissors, lit the gas in the middle of the night to keep them from smothering. He was now simple, destroyed his clothes, hid things and could not find them; his speech was at this time impaired.

*On admission* to Manhattan State Hospital December 9, 1903, the patient was quiet, childish and good-natured; he talked of having thousands in the bank (false). He did not know where he was, mistook those around him for old acquaintances. He said that it was 1893 and could not give the day of the week. On the day after admission he imagined that he had already been about a week in hospital. *Physical Status:* "Hemiplegia. Reflexes slightly exaggerated. Pupils react normally. Speech thick; tongue tremulous."

During 1904 and 1905 the patient showed no marked change. On October 14, 1905, his physical condition was as follows: A well built and well nourished man with a scar and deep depression at the site of the old osteitis of the right tibia. No external evidence of syphilis; the patient admitted some venereal infection, but could give no trustworthy information. *Slight weakness of left arm and leg;* no weakness of the face noted. Movements of the left hand were more hesitating than those of the right hand; the patient walked unsteadily and slightly dragged the left leg, which he said was stiff; the tongue was put out straight. He complained of no sensory disorder, reacted promptly to pin-pricks over face, hands, legs; he said that a touch with a brush was lighter on the right side of the face and right hand than on the left side, but his co-operation in the test was poor. The knee-jerk and the Achilles jerk were more active on the right side than on the left (? difficulty of relaxation); in fact the left Achilles jerk was not elicited (tested in bed). The triceps and wrist reflexes were active on both sides. The plantar reflex was difficult to deter-

mine as the patient kept moving his toes up and down; it appeared to be flexion on both sides. The pupils were practically equal, the left slightly irregular; the right pupil reacted promptly to light and on accommodation, the left not so promptly to light but well on accommodation; both reacted consensually. Hearing was much impaired on the left side. The speech was extremely tremulous, with omission and transposition of syllables. The writing showed the remains of good style, but marked tremor and distortion of words. The internal organs were in satisfactory condition; slight peripheral arteriosclerosis.

On November 18, 1905, the patient had a series of *general convulsions;* when seen by the charge nurse after the first convulsion (5.30 A. M.) the *left arm and leg* were twitching, as well as the eyelids on both sides. When examined at 9 A. M. the limbs on the *left* side were limper than on the right, the head and eyes were turned to the right. The knee-jerk on the left side was diminished, on the right side was active; plantar extension on both sides.

Of special interest was the patient's behavior after these convulsions; during the afternoon and evening he continually made movements with his right hand, putting up the thumb and index to the lips as if feeding himself, moving his lips and continually grinding his teeth. During the whole day he was unconscious; during the night he talked inarticulately; next day (November 19) he repeated some phrases again and again, but showed great difficulty in articulating and swallowing. He continually opened his mouth and rolled his tongue about.

After a few days the patient was in the same condition as before the attack; he showed a well marked euphoric dementia, talked of having a big farm (false), had only a hazy idea of his environment, had no idea of the date. His memory showed glaring defects, and he was quite unable to reconcile his various dates.

On May 7, 1906, the patient had a series of *convulsions.* The first three (8.10 to 8.25 A. M.) were observed by the nurse and were general, the third one beginning with conjugate deviation of the eyes and turning of the head to the left. After this attack the patient was comatose; the eyes deviated to the left; at 8.50 A. M. the *left* hand became slightly closed, and began to show clonic movements, then the left eyelid began to twitch, the eyes twitched to the left, next the left side of the face began to twitch; at this time the patient showed twitching of the left face, arm and abdomen, while the right arm and *both lower extremities* were flaccid; in two minutes the left leg became rigid, and the whole left side showed clonic movements for about two minutes. After this attack the left side of the face appeared weak, the eyes deviated to the left; the sign of Babinski was present on both sides. At 9.15 clonic contractions again began in the left hand, spread to the arm and face, and after six minutes spread to the leg. At 9.25 there was an attack in which the left arm

became rigid, then began to twitch, the face and eyes were turned to the left; the left face and arm continued to twitch for five minutes while the right arm and both legs remained flaccid. The sign of Babinski was elicited on both sides. The patient reacted very slightly to pin-pricks. The convulsions now ceased, the patient breathed deeply with blowing out of the left cheek; the left face was smoothed out, and the angle of the mouth drooped. The patient reacted slightly to sensory stimuli.

During the rest of the day the patient showed similar behavior to that which had followed the convulsions on November 18, 1905. He made continual movements with the right arm, tore his shirt with his right hand and continually put his right hand up to his mouth as if to insert something. He appeared to grip imaginary threads with his teeth, to chew them, pull them out of his mouth and roll them up; he would then rub his neck and again put his fingers up to his lips, appear to chew and draw out imaginary threads; occasionally the left hand co-operated. He did not respond to simple questions nor demands; he did not give a hand-grip.

Next day he was less stuporous and on May 9 he was in the same condition as before the convulsions; he did not realize that anything special had happened.

During the summer there was little change. On November 12 he had a series of convulsions. The first two were general; the third was general for four minutes, then the face was twisted to the left, the left arm and leg twitched more than the right. After this attack the patient returned gradually to his previous condition.

The patient died on November 30, 1906, without further neurological incidents.

CASE 2. T. R., 43, hatter; admitted to Manhattan State Hospital January 10, 1907.

The patient (a right-handed man) was born in New York in 1863, was a healthly child, developed normally, became a hatter and had a small business of his own. He was temperate in the use of alcohol; he told his family physician that when quite a youth he had contracted syphilis; nothing was known as to treatment. He married in 1892; his wife was never pregnant.

*First Attack.* In February, 1897, the patient had a transitory *weakness of the right hand* and *difficulty of speech.* When called to dinner he said "I feel awfully funny, my hand feels so numb, my head feels funny" (over occiput). He could not lift a spoon with the *right* hand; his speech was thick. The weakness of the hand did not last more than a day or two; after three days he was in his usual condition.

*Second Attack.* In November, 1897, he one day leaned to the left side as if about to fall, but was able to continue his work. Next morning his *left arm and leg* were paralyzed, his *speech* was very thick, he cried like a baby. For the next two weeks he talked foolishly, said

that the Astors were coming to steal the spoons, told his wife to send for a certain kind of hat to a Chinese restaurant; he had no ideas of greatness. He seemed to have visual hallucinations, saw acquaintances in the room who were not there; he was oriented, recognized his wife all the time. He gradually regained some power in the left side and in six weeks could walk around with assistance, dragging his left leg; at this time the left arm hung quite powerless, but gradually the weakness of the left arm disappeared until no difference was noticed between the two arms. He continued to walk with a limp.

After the second attack the patient would have little episodes of weakness, which compelled him to lie down for an hour or two; his limp usually was more marked during these spells. On one occasion (1905) he became unconscious for a few minutes.

For two years previous to admission he worried over his condition, and was rather depressed, dreaded the onset of general paralysis. In May, 1906, he walked better than usual, said that his health was coming back and soon began to show a marked grandiose trend; he said he had piles of money, talked of absurd plans. During the two months before admission he would awaken his wife at all hours to discuss his plans; he became irritable with her, and one day assaulted her. Two days later he was in a very apprehensive condition, imagined that he saw robbers entering the store, attacked his wife. He was therefore taken to Bellevue Hospital. There he was restless and excitable, muttered, sang and talked absurdly: "I got brain trouble but God I am getting better, I can run five miles—I showed my wife the revolver, but I didn't mean to shoot her—next time those Italians come into the store I will shoot them in the legs."

*On admission to Manhattan State Hospital* (January 10, 1907,) the patient was very talkative; when interviewed he answered questions freely, but was occasionally rather irritable and swore. He said that he was "as merry as the canary, happy as the day is long—I feel A No. 1." He talked of having thousands; he was going to be President; he could speak German and Italian (false); "everybody will vote for me from the anarchists down." It was difficult to know whether he had auditory hallucinations; "everybody talks about T. R. (his name)—they say he is the most religious man in the world." (Do you hear voices?) "Yes! I got all the symptoms, but I only drank beer, put it down as I am given to speak of the matter from the platform." He gave the date approximately, knew that he was in Manhattan State Hospital, but was wrong as to its situation; he gave the day of the week as Saturday (Tuesday). He gave a fair outline of his life, but made a few absurd statements and was not quite accurate in his dates.

His memory of the events leading to his admission to the hospital and of the few days in hospital was rather inaccurate. He had a fair store of general information, and did simple sums correctly. He

showed poor retention of the tests given him. He had no insight into his mental impairment.

*Physical Status:* No anatomical stigmata; no external evidence of syphilis (he admitted infection). *Slight weakness of left leg; no weakness of left arm nor face* made out. Knee-jerks exaggerated, especially on the left side. The gait was both hemiplegic and ataxic, he scraped the left foot along the floor. Sign of Romberg poorly marked. Speech hesitating and tremulous with only occasional distortion of a word ("bigrade" for brigade); tremor of tongue and face, no marked tremor of fingers. There was no gross sensory defect, but he showed only slight reaction to pin-pricks; no difference between the two sides could be made out, but he did not co-operate seriously in the tests.

Pupils equal, slightly irregular, reacted well on accommodation, slightly to light. Marked lymphocytosis of the cerebro-spinal fluid. Internal organs normal; no albuminuria.

After one month in hospital the patient showed further mental deterioration; his orientation was worse, he gave the date as June 28, 1879—"It's all summer—the moon I ordered it stopped, but the sun it is always at 5 o'clock electric light." His talk was very much more dilapidated than on admission. His account of his life contained absurd discrepancies in dates. He now had trillions; "I play with stocks—one hundred million a piece, I bet a hundred thousand dollars on the fight last week." (How do you feel?) "Very well, from my nails to my red head (brown hair) scarlet red hair—intelligent, religious, strong, union-made by God in Christ Mary Magdalen R. (his name) Amen 4 times." On April 13, his wife during her visit called attenton to the fact that his face appeared bloated; he also walked in a very lame manner. On April 14, in the forenoon his left leg seemed to give way when he was at exercise and he fell; he was unconscious for a few minutes, but was able to walk back to the ward. After dinner he was very stupid and difficult to arouse; he vomited and was then placed in bed; his temperature was 101°.

On April 15, he continued to be extremely dull, only moaned when addressed; T. 105°—P. 114; the pulse was of low tension, the pulse wave poorly sustained; the breath sounds were loud, accompanied by rhonchi. Sign of Babinski on left side. He was too dull to co-operate in examination of his motor functions. He drew up his legs when being washed. When a light was flashed in his eyes the pupils contracted promptly, but accommodation could not be excluded. On April 16, the pulse and respiration became feebler; the face was pulled to the left side; sign of Babinski was observed on the two sides; no difference of muscle tone on the two sides could be made out. The patient died at midnight.

CASE 3. J. L. W., 43, journalist, right-handed; admitted to Manhattan State Hospital, August 4, 1906.

*Family History.* The patient stated that his father, a wine mer-

chant, had some mental disorder in advanced life; his mother died of cirrhosis of the liver. No further information could be obtained.

*Personal History.* The patient gave the following information as to his life. He was born in England in 1863, developed normally, received a good education, practised as a lawyer for a short time, came to America in 1888 and worked as a journalist. Since 1894 he had been on the staff of a prominent New York paper. He drank whiskey to excess.

*In 1898* he had a *chancre* with "specific treatment" for several months (the date of infection given by the patient on admission to Manhattan State Hospital in 1906 was the same as that given on admission to the New York Hospital in 1902). In the autumn of 1901 he had *diplopia* which improved after two months treatment with potassium iodide.

*On April 21, 1902,* at lunch he was unable to use his left arm, a friend had to cut his meat for him; the leg was not affected. Next morning he staggered all over the room, telephoned for a friend who came and dressed him. They drove to a doctor who sent the patient to bed; next day he was taken to the New York Hospital.

The following is the report of the case from the New York Hospital:

"Two days before admission noticed tingling in left hand followed by paralysis of left arm and leg in twenty-four hours. *Physical examination:* Unable to draw lips back so as to show teeth; speech slow and thick. Right arm and leg normal; left absolute paralysis. Tactile and temperature sense normal. Micturition involuntary. *Treatment:* Potassium iodide. *Diagnosis:* Cerebral syphilis (left hemiplegia). Discharged May 21, 1902 (four weeks in hospital)." The patient stated that there was at that time slight residual weakness of the left arm and leg.

He was able to continue his work during the following six and one-half months; he then had an attack of which he gave on admission the following account. For ten days he had suffered from severe headache; accordingly he took a large dose of castor oil. During the night he woke up lying on his face; he could not move and was afraid he would suffocate; he managed to knock down a screen and thus attracted attention. He could not speak when the landlady looked in, and for a few hours he remained speechless (he is a right-handed man). The next day he was admitted to the New York Hospital.

Report from the New York Hospital :

"Readmitted November 8, 1902 (six and one-half months after his discharge). *History:* After returning last night dizzy and nauseated, could not rise from bed because of weakness of left leg. Clonic spasm left arm, leg and side of face—four hours duration. Not unconscious; unable to speak. Spastic paralysis left arm and leg. Patellar and plantar reflexes exaggerated. *Treatment:* Sodium iodide. Mercury bichloride per hypo. *Diagnosis:* Cerebral syphilis. Discharged, November 27, 1902, improved."

For the next few years the patient had headache from time to time, and occasionally took medicine. In the autumn of 1905, he lost his position; his work apparently was unsatisfactory. In October, 1905, he noticed that the left leg was becoming weaker; he received intragluteal injections for one month. In January, 1906, he twice (15th, 22d) fell; he had no dizziness, no loss of consciousness, he was able to raise himself and walk home.

On admission to Bellevue Hospital (January 25, 1906) he complained of progressive weakness of the left leg, loss of memory and a sense of insecurity. He remained in Bellevue Hospital until August 4, 1906. Thorough antisyphilitic treatment was adopted; he received as much as 390 grains of potassium iodide daily, and was also treated with hypodermic injections of the mercuric salts. His condition, however, did not improve. From the beginning of his stay in Bellevue Hospital he showed mental symptoms. In January he was irrational, tried to get out of bed and take a stroll along the river; in February he was restless and noisy at times, very talkative, sometimes rambling about imaginary things; on February 26, he had four convulsions; in May he seemed to have hallucinations of hearing, he had spells of wandering, tried to escape; in June he saw objects dimly, was generally muddled.

On admission to Manhattan State Hospital, August 4, 1906, he was childishly happy, affable, loquacious, amused by details, frequently laughing without much provocation. He gave no grounds for his euphoria, said that he had "not a d—d cent," and as to his physical condition he said, "it's a terrible plight—I don't suppose I will ever get well again;" he laughed cheerfully at the position.

He knew where he was, gave the date correctly. He complained spontaneously of his memory being poor, and in giving his history he made several careless mistakes; these, however, he was able to correct; e. g., he said he had been *ten* years in New York, had been working *twelve* years for the New York paper. He had a fair memory of the period since his first attack in 1901, although he said that the interval between the two admissions to the New York Hospital was eighteen months (it was six). Although his retention of a test name and number was good, he was very much confused over the incidents of the immediate past. On the day of admission he thought that he had been two nights in the hospital, and shortly after admission he confused Manhattan State Hospital with Bellevue Hospital, thought that he had been in the former several months, said that the ward physician of the former had vaccinated him at Bellevue Hospital; he was confused as to the time of day; in the evening he said, "I thought it was about eleven o'clock—I remember now I had dinner." He had some difficulty in giving simple facts; the name of the river Hudson, of the Governor of the State, etc., would escape him; the cause of the war with Spain was some difficulty over the tariff.

He did not at all realize the extent of his mental impairment, nor

that his mood was in any way abnormal. He had no morbid ideas; he admitted having had a delirious episode and hallucinations at Bellevue Hospital.

*Physical Status.* He had the well marked residuals of a left-sided hemiplegia—weakness of the left arm and leg, none of the face; left knee-jerk more exaggerated than the right; ankle clonus and sign of Babinski on the left side. No sensory disorder could be demonstrated. He was clumsy in all his movements; in walking he dragged the left leg, walked with a broad base of support, staggered from side to side. His difficulty in maintaining the erect position was increased on closing the eyes. There was tremor of hands and face, little tremor of tongue. The speech was tremulous and sticking, but without any distortion of the words even in difficult test phrases; the writing was extremely tremulous, the words were crowded up into one corner of the paper, but were correctly written. The pupils were dilated, (5–6 mm), equal, irregular; they reacted well to light and on accommodation, also consensually. No hemianopia. The radial arteries showed no marked thickening. There was no disorder of the internal organs; no albuminuria.

*On August 14*, the patient had a series of convulsions, the right side twitching more than the left. Next day he answered in a slow and dull manner; said that he was all right.

*On August 16*, he was still somewhat confused, had considerable difficulty in expressing himself, showed marked perseveration and great difficulty in getting the words he wanted. (What has happened to you?) "Oh! nothing particular—I seen you before, but I don't remember anything startling." (What place is this?) "I know I am in a *part*—a *portion*—an *apartment* belonging to the Western Union—a portion of the features - - - a portion of the apartment which I presume they want." He kept on talking about rooms, "I been letting my - - - rooms - - - I can't pronounce the words - - - I want this word in here" (finger on corner of mouth). He responded correctly to simple commands, e. g., "hold up hands," "show tongue." He was able to name some objects correctly but showed marked perseveration and occasional paraphasia. (Pencil) "Pencil—Harlem—alum." (Watch) "Pencil" (Keys) "Pencil aint it?" (Blanket) He reached for it and fingered it, "I wasn't to feel of it—a piece of silverware." (Quarter) No response. (Silver knife with mother-of-pearl handle). "It looks like a silver coin, I can't see it"—"That's a silver knife—that's the only one I guess right." The *right* arm was weaker than the left. He could not hold the pencil well enough to write. He continually fumbled about his face and chest with his hands in an awkward manner.

*On August 17*, he was brighter, named objects correctly without any perseveration; he readily understood spoken commands. He was given a letter to read, he held it awkwardly, fumbled and dropped it frequently. He read his own name quite incorrectly; (John L.

W.) "Nother or mother—mother—thodack—don't know which it is—John L. Nothon (What?) Oh! I said it was Thodack." The letter began: "Dear John I am glad to hear you like your new quarters and am sure it will do you much more good than being in Bellevue;" he read, "Dunnell W.—you like your new quarter—New York City very much—very much you—glad—you Bellevue, you like to be in Bellevue."

On August 16 and 17, he said that he saw less distinctly on the right side than on the left.

*On August 18*, without any convulsion having been observed, the aphasic symptoms were very marked; he was drowsy in the morning, but when interviewed became brighter, tried to talk; he uttered senseless jargon or reiterations, with only an occasional intelligible word. He said spontaneously, "No jection—dab—jection—debjection." (What is your name?) "Debjection (laughs) that's right—debjection—la-jection—the objection—I objection—debjection—now objection - - - - muligal—musge sell—wood—good—sell - - ."

He made few responses to commands, paid little attention to objects shown, gave irrelevant answers: e. g. (what is your name?) "Yes! I'll go." (Q. repeated) "I think—think I'll—I'll—."

The *right* arm, which had been weaker than the left since August 14, was still further affected, lay across the chest and twitched from time to time; he gave no grip with the right hand. He moved both legs readily in bed. The reaction of the pupils was uncertain; hemianopia not demonstrated, co-operation poor. The right face and arm were less sensitive to pin-pricks than the left, but the right leg was more sensitive than the left which was very dull. Knee-jerks, both increased; ankle-clonus and sign of Babinski on the left side.

During the next few days the aphasic symptoms became more pronounced, the patient became duller; he gave no evidence of understanding anything said to him except an occasional response to a command (raise arms! show tongue!); he poured out a stream of gibberish. The pupils reacted very slightly to a strong light. The right arm remained weaker than the left, and showed some twitching.

*On August 22*, he was brighter, carried out correctly a few spoken commands, named a pencil correctly after several paraphasic attempts; he showed marked perseveration. The improvement was only temporary. On August 25, the patient passed into a semicomatose condition from which he could not be roused; the eyeballs twitched, tended to roll to the right, there was a tendency to turn the face to the right. The limbs were all flexed except the right arm which showed some rigidity and coarse, jerky movements; there was considerable twitching of the lip muscles. On August 26, the patient died.

CASE 4. W. C., 47, admitted to Manhattan State Hospital, August 25, 1905.

*Family History.* The father was alcoholic. The mother married at 16 and one year later had twins, of whom one was the patient.

*Personal History.* The patient was born in New York in 1858; he was a neurotic child, had night terrors, frequently walked and talked in his sleep. He was a dull scholar, but a bright comrade. He followed a variety of occupations, became addicted to alcohol, married in 1891, having had a chancre six months previously for which he received only three months treatment. He infected his wife and she had a disastrous series of five pregnancies, but finally a living and healthy child. Even after marriage the patient continued to drink immoderately, indulged in excessive sexual intercourse, tormented his wife with his morbid jealousy and suspicions, so that she left him more than once.

As far back as 1897, he would suspect poison, and imagine that his relatives and associates dealt unfairly with him; his waking suspicions were apparently considerably influenced by dreams. It was with difficulty that he could be brought to realize that he had merely dreamt that an acquaintance and his wife were plotting to kill him. For a considerable number of years he had an easy post as janitor of a large office building. In 1900, during the summer, he was very much exhausted; he sometimes appeared about to faint. Towards the end of the summer squint developed and persisted until November. *In September* he had an attack of dysarthria and staggering, but no special weakness of the limbs of either side was noticed by his wife. He recovered after two months in bed, his speech returning gradually to its normal condition. In *1901*, he again became fatigued, lost appetite, and *in September* he lost the power of his *right* leg "from the knee downwards" (wife's account); he could not stand on that leg. He did not "mow" with the leg in walking, but the leg seemed to dangle from the knee. This weakness did not come on abruptly, but in the course of a few days. His speech was a little affected; the arm showed no special weakness.

For the next two years (1901–1903) he was unable to work; he dragged his right leg when walking, on some days more than on other days; he had two or three sudden attacks during which the right arm would be completely paralyzed for half a day, while the whole right side would be weaker for about a week, and the speech remain affected still longer.

He knew that he was an invalid, but took an optimistic view of recovery.

In *1903*, one day he suddenly fell; when seen one hour later he was a little excited, and able to speak; his *left* arm (his wife insisted that it was his left, and the patient talked of one left-sided attack) became paralyzed, and he could not speak for an hour; at the end of this time he was able to walk up three flights of stairs, and showed no additional difficulty of articulation.

From 1903–1905, he was a night-watchman; although he was becom-

ing gradually weaker he made light of his hemiplegia. He had occasional difficulty in controlling his urine.

About the end of July, 1905, one day he was found unconscious in bed; in half an hour he was found sitting on a chair, he was much excited, tried to speak but could not; at first he made signs, then after a few minutes said "that's better," and became unconscious again. For about a week he was semi-conscious, had no control of his organic reflexes; during the following month he remained at home but was delirious, thought he was on a ship, saw strange men, for a few days he did not recognize his wife.

He refused food and medicine because it was poisoned, and was taken to Bellevue Hospital. There he thought that he was in church; he appeared demented and childish, had "ill-defined delusions of grandeur and of persecution."

*On admission* to Manhattan State Hospital (August 25, 1905), at first he was excited and pugnacious, but on examination became quiet and agreeable and was able to maintain a conversation satisfactorily; he was rather amused and commented on the details of the examination. He was happy, felt first-rate, did not resent being with crazy people. He gave the date correctly; "This is Manhattan Life Insurance—Bellevue Hospital." He gave rather a poor outline of his life with marked discrepancies in dates, e. g., "This is 1906—I was born in 1862—I am 47 years old." His general knowledge showed considerable dilapidation. He had no adequate realization of his general impairment.

*Physical Status.* Right hemiplegia; defective speech; pupils reacted well to light.

The patient continued to present the same general picture of mild dementia for the next few months; he whimpered easily over detention, but on the whole showed rather excessive good humor and complacency; his memory for recent incidents was very poor, he talked of his wife having called yesterday when it was his mother who had called two days previously. On October 25, left-sided ptosis developed and became complete in a week; it cleared up after four weeks.

*Physical Status* (October 25, 1905). A well built man with no anatomical stigmata; scar on penis and in groin; weakness of the whole right side—face, arm, leg. Knee-jerks and Achilles-jerks exaggerated, especially the right. The gait was ataxic as well as "mowing." Babinski reflex and ankle clonus on *both* sides. Slight ptosis on the left side; the left pupil was widely dilated, did not react to light nor on accommodation; the right pupil was smaller, reacted slightly to light, well on accommodation. The fundi were normal. No hemianopia. Speech was slurring, slightly sticking, without omissions or transpositions, but with the occasional insertion of r; the writing was tremulous with omissions and distortions, e. g., "methis espical" (methodist episcopal), "bittililery" (artillery). There was no gross

sensory disorder. The cerebro-spinal fluid showed a very marked lymphocytosis.

The patient continued during the following months to present the same picture of mild general dilapidation, with no adequate realization of the mental and of the degree of the physical impairment, but without showing any definite expansiveness or other abnormal mental trend while in hospital. The left pupil continued to show no reaction to light, but it slowly came down somewhat in size.

The diagnosis of general paralysis in this case was not considered to be definitely established, for the neurological history with its varied incidents seemed to point equally in the direction of brain syphilis. An attempt was therefore made to give as much definition as possible to the elements of the dementia. The mood continued to be one of rather exaggerated complacency with a tendency to whimper when talking of home; he was anxious to go home. He admitted that he was weak, but thought that he might resume his old work; he had been able, even when hemiplegic, to work as night watchman, and he said that he had good recommendations; there was nothing intrinsically absurd in his plans for the future.

His memory was rather poor and he showed great difficulty in handling dates, and in holding the various statements together.

*December 23, 1905.* (How old?) "47." (When born?) "1857 (after cogitating)—48—1848—I'm 57." (Where born?) "I was born in 1848 I think." (How old?) "47." (1848+47?) "That would leave me—I would be 48 instead of 47, 48 I should say my age is." The examination of any similar point was apt to lead to absurd and contradictory statements. He showed considerable pertinacity in trying to reconcile some of these statements.

He realized keenly how dishonorably he had acted in infecting his wife; he still retained appreciation of social differences, e. g., (Your speech is pretty bad) "Mine ain't excellent at all, but mine is a little refined towards those people" (*i. e.*, in comparison with the other patients). He was much pleased at being demonstrated in the physician's private room to some other physicians. He occasionally had some difficulty in finding words to express himself, e. g., on April 16, he said that he was about to have another attack, "I have the *pheazness*, the same *systems*, I have the same touch on me that I had before." He complained of feeling dull and faint in the head. No attack occurred at this date.

On April 30, 1906, after having on the previous evening shown some confusion, the patient had a general convulsion early in the morning; both sides seemed to be equally affected. During the whole day he was unconscious; the right arm was held either against the side, or in the wing position, while the left arm moved about restlessly. The mouth was pulled to the left. Ankle clonus and sign of Babinski on both sides. The right pupil was larger than the left (the reverse of what had hitherto been the condition).

The patient did not come out of his stupor and died on May 3.

CASE 5. M. D. R., 58, admitted to Manhattan State Hospital, July 12, 1905.

*Family History.* The grandparents and parents showed no neuropathic nor psychopathic traits. The patient was the third of a family of eight children. One brother, nine months younger than the patient, began at the age of 45 to have fainting spells; "fatty degeneration of the heart" was diagnosed; in 1894 he had an apoplectiform stroke, and over a year later he had a second. His memory later failed, sometimes he became confused and did not know where he was. There was no history of his having had syphilis, but his wife had never been pregnant.

*Personal History.* The patient was born in 1847 in Heligoland; he was born at the seventh month, developed slowly as a child, and was rather below the average at school. He came to the United States when 15, followed various occupations, was in the navy from the age of 21 to 24, and after that time he worked as a barber in New York. He had been fairly temperate in the use of alcohol. In the navy he received some venereal infection, "a soft chancre—chancre wart—several of them," which disappeared in two weeks and was followed by no secondary symptoms. About 1890 the patient had severe hemorrhage from a varicose ulcer, required transfusion, spent several months in hospital; no record of his condition at that time could be obtained. In 1893 his mother died and even at this date the patient was rather forgetful; it was, however, not till 1902 or 1903 that he showed definite signs of mental impairment. About 1902 he was noticed to be absent-minded and very forgetful, unable to remember the visit of a friend soon after it was made. He stayed at home, partly on account of a troublesome varicose ulcer, and for the three years previous to admission had done no work. His memory became progressively worse; he showed no abnormal mental trend, was quiet, affable, compliant.

In the first week of July, 1905, he one day started to leave the house although only partially dressed, he was dazed, could not explain his conduct; half an hour later he had a general *epileptiform convulsion* and was immediately taken to Bellevue Hospital.

In Bellevue Hospital he was restless and confused, disoriented, and with extremely poor memory of both recent and remote events; "I came here last October—this is February—I think it is 1875 or 1879—I came to have my leg dressed—I am feeling first class—I am as strong as an ox—I can't do much work of late—I am getting old and weak." His mood alternated between elation and irritable depression.

He was admitted to Manhattan State Hospital July 12, 1905.

*On admission* the patient showed little interest in his new environment, assisted in undressing and bathing himself; in bed he was somewhat restless and fumbled with the bed clothes. When examined he was rather inattentive, but answered questions relevantly; he gave his name and occupation and said that he knew he was in a

hospital; "I was sick, but I'm all right now—I got dizzy and was taken to the hospital." "I feel first rate—I ain't got no sadness at all—I am strong and healthy now." There was no evidence of hallucinations or delusions. He was not quite clear as to the place, called it Bellevue Hospital (Manhattan State Hospital); gave the date as June, 1885, the season as Spring. He gave a fair general account of his life, but wrong dates, with discrepancies which he did not realize; he was born in 1845, his present age was 52, the present year was 1885; he said that he had a chancre in 1860 (really about 1870).

He had poor memory of the events of the immediate past; "I was in my room yesterday" (false), but remembered in a confused way what had happened; "I was brought here by an ambulance—I was unconscious when I was taken." He did not remember when he had come to the hospital, although it was on the same day as the examination; he could give no account of how he had spent the day. He could not retain even for ten minutes the name of physician, a given number, or color. He answered fairly well simple questions on general topics, but said that the President was Hayes (Roosevelt); he was able to count up 90 cents correctly, grasped the meaning of a short paragraph which he read, wrote his name very poorly, the word Rust being an illegible and tremulous scrawl, one letter being written apparently on the top of the other, and several attempts having been made at the same letter.

*Physical Condition (July 14, 1905).* The patient was a poorly nourished man of slight build, height 5 ft. 4½ in., weight 123 lbs.; his ears were flattened and projected prominently; he was able to move them freely. He explained their appearance as due to their having been frozen 30 years previously (false explanation). There was an extremely extensive scar from previous varicose ulcers over the left shin. There was no external evidence of syphilis. There was moderate thickening of the radial and temporal arteries; the temporal was somewhat tortuous; marked thickening of brachial. There was no disorder of the cranial nerves. The pupils were noted as equal and regular and reacting normally; four days previously another physician had noted them as unequal and not reacting to light (?). No hemianopia. The knee-jerk and Achilles-jerk were not elicited on either side; on both sides the plantar reflex was flexion. No muscular weakness was observed, but the patient was rather unsteady in the upright posture, he swayed from side to side when walking; closure of the eyes made little difference; there was no tremor of tongue nor fingers. The speech was very indistinct, this being attributed to want of teeth; it was not tremulous; the writing, as noted above, was tremulous and some words were so imperfectly written as to be illegible. On July 14, however, he wrote his name legibly, while two days previously the word Rust was illegible. No gross disorder of sensibility was observed; touch seemed somewhat impaired.

The heart was slightly enlarged but otherwise there was nothing to note in the condition of the internal organs. The urine was of good color, S. G. 1017, contained no abnormal constituents. The patient had no difficulty with micturition, had control of his organic reflexes.

During August he showed no change, would lose himself in the ward, had difficulty in finding his bed at night.

*On September 3, 1905,* he became weak, dull and stupid, complained of his stomach; temperature 100°, pulse rapid. At 11.45 A. M. he had an attack; the eyes were drawn up and to the left; twitching of the facial muscles most marked on the left side; rigid contraction of the left arm and leg. He had several attacks of twitching but they were not severe; he did not entirely lose consciousness, was confused. In the intervals between the spasms he clasped his head with both hands as if in pain. He clutched convulsively at any object before his eyes. It was noted that his right pupil was contracted. The attacks ceased at 12.30, but at 1 P. M. he had another similar attack lasting three or four minutes, without loss of consciousness. In the evening the patient required to be catheterized; the urine contained a considerable quantity of albumin.

*September 4.* The patient remembered having yesterday had severe headache and attacks without loss of consciouness. Right pupil is contracted, the left slightly dilated.

*September 8.* "I feel numb in the head – – I have trouble in getting things right—I am confused."

*October 2.* Twitching during the night; in the morning two general convulsions, each lasting about two minutes.

*October 3.* Dazed, confused, tries to get out of bed; sees imaginary objects on the wall, "is practically inaccessible mentally."

Patient improved and soon was out of bed. His pupils at this time were noted as reacting sluggishly to light; his speech was thick; left side of face flatter than the right. His mental condition was practically the same as on admission.

*November 5.* General convulsion of a mild form this morning; subsequently he was confused and had no clear idea of his surroundings.

During November he occasionally required to be catheterized.

*November 21.* "Series of convulsions of a mild nature followed by confusion and restlessness." Cerebro-spinal fluid shows definite lymphocytosis—30 lymphocytes in one field (oil-immersion).

*November 22.* "Mild attack with twitching of arms and hands."

*November 28.* After a few days he was in the same condition as before the attacks. He realizes that he is in a hospital for the insane, does not think he is insane, says he has fits, was out of his mind when he came.

The patient's mental condition showed no change during the next few months, but he had occasional attacks with episodes of restless confusion; after one attack he called loudly for help, said that he was choking; he fumbled along the wall as if groping for imaginary objects.

*March 23, 1906.* This morning after being shaved the patient was seen groping along the corridor; his head was turned to the right, his eyes moved uneasily to the right. His left hand was very weak and he held it out in an extremely tremulous and ataxic manner; tactile sensibility was impaired on the whole left side; left-sided astereognosis. When examined in bed it was noticed that he saw better in the right visual field than in the left, and during the examination this hemianopic disturbance passed into general blindness. At this time he was lying with eyes turned to the left, the left hand showing almost continual restless, jerky movements.

From the beginning of the examination the patient was in a quasi-delirious condition, and talked in an extremely tremulous and almost inarticulate manner. He asked for water: "I have to swallow it—I've been told every time I've come here—you've the bon—I want a comb." He stretched out his hand; "I am only looking for the rope that's running round." He knew he was in a hospital. After some time he lay groaning in anxious distress—"Oh, oh, fire—stove, aint it—aint you."

In the evening he had six convulsions of short duration; one was reported as being right-sided.

*March 24.* After a restless night the patient was in the same confused, distressed condition as on the previous day; he mixed up delirious utterances and correct references to physician and hospital. He was still quite blind, could not grasp a hand held out, nor point to the window. At meal-times it was necessary to put the cup into his hand. When asked to point out the doctor, he pointed vaguely upwards and around, said "I can't see for the shine." He sat up and fumbled; "there's a door between you." He named correctly objects in the right hand, still showed astereognosis on the left. He grabbed his own left hand with his right, as if it were a foreign body, said "there's your finger," and with his right hand he opened and fumbled the fingers of his left hand.

*March 25.* The patient slept well after chloral hydr. gr. 15, pot. brom. gr. 20. In the morning he was still blind, unable to see a cup handed to him. At dinner-time he was able to see a cup, and in the afternoon he was mentally and physically in the condition preceding the attack. No limitation of the field of vision was noticed; he promptly grasped without fumbling a hand held out either to the extreme right or left. Hand grip equal on the two sides; no astereognosis. He had no memory of the attack; to explain his being in bed he said "I had some sickness in my head—I fell down in the street a couple of days ago—I was taken to the station house—I didn't know where I was, and then I suppose I was taken to the hospital."

*April 3.* The *mental status* is practically the same as on admission. He has a confused grasp of the enviroment, mixes it up with the other hospital where he was. He gives a rather confused account of his own past; he remembers the events of his life fairly well, but

mixes up the dates and sequence of events. He has a very poor idea of the period spent in hospital. He answers well simple historical and geographical questions, but has no knowledge of modern political leaders. His mood is rather depressed, but he occasionally chuckles fatuously and shows an inclination to jest. He recognizes his memory defect—"I am very short of memory that way eh ! forget everything lately - - - - I am worrying and worrying—I am disgusted with life, I ought to be dead." He has some realization of his general impairment. (Why are you here?) "Well I don't know—I suppose the people think I ain't capable of taking care of myself . . . I could work in the barber trade." (Should you have been sent here?) "I believe so—I think so." (Was it right to send you here?) "No, sir—it was not. It was right to send me here to be cured of the spell."

*Physical Status.* Knee-jerks and Achilles-jerks absent; plantar reflexes flexion; left pupil irregular, larger than right, both pupils react to light and on accommodation, the left a little more sluggishly than the right. Hemianopia not made out—the right side is a little doubtful (patient is rather stupid). No motor weakness; no sensory disorder, but co-operation is poor. Tremor of hands and facial muscles. Speech extremely defective with tremor, sticking and transpositions. Gait unsteady, not definitely ataxic; well-marked sign of Romberg. He has various subjective feelings - - "my head aches—the same as a drum inside;" he is very dizzy, and for a short period looks dazed, sits with head sunk forward. During mental examination he said, "I've got a pain in my head all at once - - - I am *black in part of the eyes.*"

*April 4. Right-sided hemianopia.* This morning he is roving about as if blind; is put to bed. In the afternoon he takes a cup promptly when held to his left side, *but not when held to the right.* He is generally confused, misnames objects.

Next day (April 5) the patient was confused, but knew physician and that he was in a hospital. He made confused semi-delirious remarks without any context, e. g., "look out! you will break that." He called a half dollar a dime, reached for it with his mouth.

After a few days he was again clear and was allowed out of bed; whether the right-sided hemianopia persisted or not, is not noted.

*May 10.* The patient had an attack during the night; showed no residual in the morning, did not remember the attack.

*May 19.* To-day the patient suddenly became blind, was put to bed. His head and eyes were directed to the right side, the eyes never coming to the left of the middle line. Trace of albumin in urine. He was mildly delirious.

*May 20.* At 11 A.M. he was still completely blind with eyes never brought to the left of the middle line. At noon he moved his eyes fairly well in all directions, and could see in the left visual field, although rather indistinctly; the right-sided hemianopia persisted He said, "My head aches like the deuce."

*May 21.* To-day the patient had an episode of ten minutes duration during which he fought vigorously with an imaginary man who was choking him; he looked sharply *to the right*, but occasionally to the left. "What have I done to him—what the deuce have I done to that man? They are choking me." He said that it was his brother-in-law and another man, "I heard him sneak up upon me." He was able to count correctly the number of fingers held up.

*May 22.* The patient could not be induced to write, nor copy writing; he copied simple outlines correctly.

*May 24.* Perimetric examination shows right-sided hemianopia, the macula not being included in the blind area. The same result was obtained four days later.

*May 31.* This forenoon the patient suddenly became completely blind; he could not grip at all with the left hand, left leg weaker than the right (no motor disorder on May 30); almost complete anesthesia and analgesia on the left side of the body—face, conjunctiva, arm, fingers, trunk and leg; no sign of Babinski. Left arm more ataxic than right arm.

In the afternoon he lay in bed with head and eyes turned to the right; the eyes rarely passed the middle line to the left. He was able to give some grip with left hand, pushed blindly forward with left arm, was able to walk with support. Sensibility—touch, pain, localization—was still defective on the left side. He seemed to have a vague perception of the light from the window. He treated his own left hand as a foreign body—"here's your hand—here's the thumb—here's the forefinger."

*June 1.* Physical condition is practically the same as on the previous afternoon; *right* hemianopia, weakness and ataxia of *left* side, defective sensibility of left side; no loss of muscular sense; well marked sign of Romberg; no speech defect noticed.

After the physical examination he had a quasi-delirious episode, fumbled about in great distress—"I can't get up—will somebody go! Oh, God! I can't run across the street in my nakedness - - - I am senseless, I dropped down and my sister died and I—Oh, God, she poisoned herself—oh! oh! oh!" He seemed to have excruciating pain in the head, not definitely localized in any area; "I have got such a headache—my head is almost busted." At the same time he knew he was in Manhattan State Hospital, was accessible, gave his name correctly.

During the first half of June the patient had no attacks; he occasionally talked in a delirious manner.

*June 20.* In the forenoon the patient was examined, showed neither motor weakness nor sensory defect; he showed the usual right-sided hemianopia. At dinner time he said "you'll have to feed me, I'm losing my sight." It was now impossible to get him to turn his eyes to the left of the middle line. (Look at your elbow!) "I can't—I'm color blind." There was marked facial twitching, much more marked

on the left side; there was jerking of the left hand which was synchronous with the twitching of the left side of the face. He grasped the left hand with his right, as if he were grasping a foreign body. (Whose fingers have you?) "Mulligan's." (Not yours?) "Certainly not—these are mine" (holding up right hand). Occasionally he chuckled fatuously. In the afternoon he was extremely restless, fell out of bed, laughed and made light of it.

In the evening he lay in bed groaning and grunting, was inaccessible, reacted with confused distressed reactions to pinches on either leg; no motor weakness was observed. Later the attendant noticed generalized twitching.

*June 21.* The patient is still quite blind and confused. His left clavicle is found to be fractured due to the fall yesterday; treated by Sayre's method.

On the evening of June 22, the patient was able to see in the left visual field. Definite right-sided hemianopia.

For a month he showed little change, was confined to bed owing to the fractured clavicle; he con not explain why he is bandaged, says that he fell out of the window, his right hand is full of glue (it is also bandaged); he talks in a jocose manner.

*July 15.* At 7 P. M. the left side of the face was seen to be twitching, the left arm was twitching, the eyes were turned to the right, the patient was completely blind. He treated his left hand as if it were a foreign body, caught hold of it, said "here's a hand." The left arm was almost completely anesthetic, only one doubtful reaction being obtained; later tests produced vague reactions of distress when the left finger tips were pricked; sensibility over the whole left side except over the face diminished. While the sensibility was being tested the left eye began to shut and open convulsively, the left hand began to twitch, especially the adductors of thumb, then twitching of left face, then the right eye began to shut and open, and the right side of face to twitch. During all this time the patient was quite accessible, but talked in a quasi-delirious manner, mistaking physician for his brother-in-law (a frequent mistake with him).

*On July 16*, he was able to follow objects moved in the left visual field, but saw them indistinctly; he called a watch "some silver thing," a boot (tan) "a pair of gloves," he guessed incorrectly the number of fingers held up. Even in the evening he called a flowerpot "a drinking cup," but recognized it as soon as he touched it.

*On July 17*, he had returned to the condition preceding the attack, saw and named well objects in the left visual field. He still made delirious remarks—"the milk is running through the wall;" but at the same time had some grasp of enviroment . . . "in a hospital and two doctors" (correct). He chuckled good-naturedly without any special ground.

*On August 4*, he had a confused episode with vivid auditory hallu-

cinations, he ran hurriedly into the dormitory apparently in answer to a voice; when made to sit down, he soon jumped up: "He is calling me—Rust," ran into a room, asked a patient what he wanted.

*On August 26*, he had a convulsion early in the morning with residual weakness of the left arm, which lasted for a day or two.

*September 8.* Patient has a severe conjunctivitis which is treated with frequent douching with boric solution.

*September 10.* At 10.30 A. M. when examined he seemed as usual. At 11.30 A. M. he is sitting with the left arm held rigidly in the wing position, the hand blue and somewhat edematous; he is totally blind.

*At 1.35 p.m.*, the left hand was noticed to be continually flexing abruptly with twitching of the various fingers; the left eyelid also twitching. He was able to open slowly the fingers of the left hand, the fingers jerking all the time. The left side of face soon began to twitch. He did not give any hand-grip with the left hand. He put the right hand over the left hand to stop the twitching, and later rubbed the left hand. At *1.52*, the left shoulder, arm and forearm were all violently jerking while the fingers were quiet. The legs were not implicated and showed no motor weakness. When the sensibility was tested at 2.32 he did not react at all or only vaguely to pin-pricks over the left hand and arm, but reacted to pricks over the left face and leg and the whole right side. At *3.25*, chloral hyd. gr. 20, pot. brom. gr. 30, were given and the twitching soon ceased; the left arm remained very rigid. He continued to make delirious remarks and was restless.

*On September 11*, sensibility had returned to the left arm, he reacted promptly to pin-pricks all over; he was able to follow objects with his eye, but did not seem to grasp their nature. He gave a rather weak grip with the left hand, "this is a little lame," he rubbed it with his right hand.

*September 12.* He still had difficulty in naming objects seen in the left visual field.

*September 13.* He could name quite well objects shown in the left field; was generally clearer.

The patient continued in the same state for a month, was able to go about the ward.

*On October 12*, he was seen wandering round the ward in a confused way; he was unable to see anything, was put to bed. He lay in bed groaning in great distress; he grasped well with the right hand; he gave no grasp with the left hand, which was slightly cramped. He rubbed the left arm in a distressed manner. Sensibility on the right side was intact; on the left side it was generally impaired, but not uniformly. On the left side he reacted although imperfectly to pin-pricks over the face; vigorously to pricks over the finger-tips; he did not react to pricks over the forearm, but somewhat over arm and shoulder; he reacted well over the left trunk; imper-

fectly over the left leg (in the previous attack on September 10, sensibility over the left leg was not impaired).

*The attack like the previous one consisted essentially in mildly delirious behavior, in temporary elimination of the left visual field, weakness of the left arm, and impairment of sensibility on the whole left side.*

*October 13.* In the morning the left hand was still numb, but no defect in sensibility could objectively be determined; he was blind. At noon he had some visual perception; in the evening he saw quite well in the left visual field.

During the following night he was much excited, talked of there being snakes in his bed, he bit his own arm. For the next two weeks the patient remained at about his usual level.

*October 24.* The patient had a general convulsion preceded by a variety of twitchings beginning on the right side; the whole episode lasted from 10 to 15 minutes. The following was the series of events: At 11.45 A. M., when summoned for dinner, he made no response, stood looking in front of him; the eyeballs showed nystagmoid movements towards the right side, he nearly fell but regained equilibrium, right thumb began to twitch, then some of the other fingers; he reacted less to pin-pricks on *right* side than the left. When asked to put out his tongue, he sniggered, made no response. Right orbicular began to twitch, then left orbicular, flickering of corner of mouth, head pulled round to right (is placed on a chair with difficulty as he does not unbend), right frontalis contracted, fingers were now twitching vigorously, left angle of mouth was continually jerked down, elevators of right angle of mouth twitched vigorously. He now uttered a cry, mouth was pulled to right, legs raised stiffly off chair, he became livid; after a short time wide clonic movements of legs, then sudden relaxation, deep stertorous breathing. The last twitches were in left hand.

In the afternoon he had two more convulsions; was later restless, distressed, moved his hands freely but handled the left with the right as if it were a foreign body. Pin-pricks on the left hand were not reacted to; were reacted to on *right*.

*October 25.* In the morning he was quite blind, but said that he could see. In the afternoon he said that he could not see, but evidently had some visual perception, grasped with his hand objects held in the left visual field, followed them with his eyes, but did not recognize them. He evidently experienced some difficulty in visual perception—"you're half a mile away—farther off." Sensibility of left arm showed no defect.

*October 26.* Sees well in the left visual field; shows his usual mild confusion and poor orientation.

*On November 9,* he had another attack with obliteration of the left visual field. There was twitching of the left face, platysma, orbicularis palpebrarum; the eyes were almost always turned to the right.

No reaction to pin-pricks on left arm, face, leg; accurate reaction on the right.

*November 10.* Continues in the same condition, blind and confused.

*November 11.* He followed objects in the left visual field, named correctly some objects shown, added delirious statements, e. g., "I saw the cat running past too—the poor dumb an - - animal." The left eye was less widely open than the right.

He did not react to pin-pricks on the left side except over forehead. The left hand grip was loose and clumsy. He was jocose, made foolish remarks with no context, said his left arm was pasted on, he could steal a penny off a dead man. When visited by his sister, at first he did not recognize her.

*November 12.* The blindness on the left side has passed away; he says himself—"I can see better." There is no anesthesia on the left side. He continues to make delirious remarks.

*December 3.* For the last ten days the patient had been wandering rather disconsolately about the ward; he sometimes said that he had been put out of his house. This morning he became blind, was placed in bed. Pin-pricks on the left side—face, arm, trunk, leg provoked no response; he chafed the left with the right hand. He gave a very feeble grip with the left hand, grasped vigorously with the right. He walked with short, uncertain steps, and on the outside of the left foot.

During the following night and next morning he had a series of convulsions; on December 4, he gave no sign of recognition, was quite irresponsive, in the evening had two convulsive attacks, and spent a restless night.

In the morning (December 5) both his arms were jerking; there were clonic contractions of the shoulder muscles, the fingers were not twitching; the right leg was occasionally moved, the left leg lay quite still. Sign of Babinski on the *right* side; plantar flexion on the left. Although the left leg was quiet it was very rigid and the muscles were seen to contract from time to time. The head and eyes were turned to the right, the mouth drooped to the left and was wide open; the right side of the face twitched occasionally.

The movements continued from early in the morning until ten o'clock when they ceased for a short time. He did not react to pin-pricks over the left side, finger-tips, arm, shoulder, face, except over left jaw; he reacted over the right finger-tips and over the right face, but not to pricks over the right arm and shoulder. It was not possible to determine the sensibility over the legs.

The movements continued all day, and the patient had two convulsions in the evening with marked throwing about of the limbs, especially the right leg and left arm (attendant's note). To protect his right leg the bedboard had to be padded.

*December 6.* Convulsion at 5 A. M. When examined at 10.30 A. M. he was quiet but soon began to show restless movements of both

hands and fingers; the fingers of both hands flexed spasmodically and simultaneously. At first the hands alone moved; then the arms began to jerk irregularly. Sign of Babinski on the right side; flexion on the left side.

The movements continued during the whole day, but in the evening they were less limited to single muscle groups, but consisted in rather aimless abrupt movements of the whole limb. He was slightly brighter than in the morning.

*On December 7*, he was cheerful and talkative in the morning; at 8 A. M. was dull and irresponsive; at 11.15 A. M. he was brighter and talked in an unintelligible, indistinct manner. He did not utter well-pronounced distorted syllables, but at first a mere washed-out imitation of speech in which no word nor syllable could be made out. Later his utterances were more distinct. He was mildly delirious—"Kni—knife—kni—never carry—I never carried a knife since I stabbed that fellow—since I carried—since I stabbed that fellow." He did not recognize his sister. The left arm was somewhat restless; with the right arm he made incomplete quasi-purposeful movements, which began as if with some definite aim and ended in quite aimless restless movements.

The right leg was limper than the left and showed the sign of Babinski.

*From December 7 to December 15*, the patient remained restless and mildly delirious; was apparently completely blind. He appeared to hear things—"there's the bell," picked imaginary things off the bedspread. The left hand took *less* part in the delirious movements than the right. He sometimes suddenly acted on the defensive—"don't be hitting me on the nose" (no one was touching him). He was apparently blind until December 15.

Sign of Babinski on the right side persisted, was noted on December 12.

*On December 15*, in the forenoon, the patient was able to see in the left visual field; he adopted a sparring attitude to physician, did not speak even when coaxed. Plantar reflex on right side;—flexion. In the afternoon he appeared to be quite blind, was delirious and distressed.

*December 16.* Sight had returned in the left visual field. The patient was still mildly delirious, but recognized his sister.

*December 19.* Ophthalmoscopic examination under atropine (Dr. Holden): Right disc normal; left disc not seen but vessels of fundus normal. He counted correctly fingers held up.

*December 23.* The patient was able to see in the left visual field, read the time correctly, but during a visit he had a little episode of incoherent excitement, in which it was doubtful whether he could see; later he was able to grasp objects in the left field.

*December 31.* The patient has continued at the same mental level, just bordering on a delirium; he fumbles about in bed, mutters frag-

mentary remarks in German and English. He has some visual perception, sees a lighted match in the left field; he is frequently difficult to examine.

*4 p. m.* He appears delirious, making movements with the right arm as if throwing, picking up objects; the left arm makes jerky movements of less extent. Reaction to pin-pricks was everywhere fair except over the *left arm and hand.*

Sign of Babinski on the right; brisk flexion on the left. No weakness of either hand grip. Both pupils react to light; the right is larger than the left; both are irregular.

*January 1, 1907.* The patient to-day names correctly objects in the left visual field. Lumbar puncture discloses abundant lymphocytosis of the cerebro-spinal fluid.

*January 3.* Mildly delirious, talks of being in a barber's shop (occupation delirium), picks imaginary threads off fingers. He sees in the left visual field. Do you know Dr. C. (his physician)? "He's situated Ward's Island (puts out his hand), this is sick bay Island of Manhattan." Do you know Dr. C.? "He's acting doctor on the isl—of—island of Ward's Island—he's in the sick bay of the island of Manhattan."

During January the mental level of the patient varied from time to time; he was frequently rather difficult to rouse; he frequently showed mild occupation delirium. When interviewed in the middle of a mild occupation delirium he could be roused to show a fair grasp of the environment—"Oh, is that you doctor? I was going to make my bed" (has been fumbling about). "I'm sick—I'm in the doctor's care—I know I'm very sick," "memory's all right." "This is a broker—room—a boarding house." He was able to see in the left visual field, but gave evidence that occasionally in the restricted field vision was indistinct, *e. g.*, he once said "all that I see is the light" (electric lamp), and was unable to name a coin. He was able, however, when roused, to recognize printed letters, he read U. S. as "uns," and Rust as "Roost—rust—what you get on a stove." The sign of Babinski persisted on the right side; occasionally he grasped his left hand in his right as if it felt different from the right. The pupils reacted somewhat sluggishly to light, the left more sluggishly than the right.

*February 1.* The patient is very feeble, he can not be roused at all. Pulse 56; heart sounds clear; no respiratory nor abdominal symptoms. His excretion of urine has been very variable in amount; he has frequently required to be catheterized.

During February the patient showed little change, but lay in bed, fumbling and groping from time to time. At times he reacted to objects in the left field, at times he appeared quite blind; the pupils reacted slowly and slightly to an electric lamp. Occasionally he made delirious remarks.

Early in March the right side showed clonic contractions and on

March 25 there was twitching of the right face, arm and leg with no reaction to pin-pricks over these regions, while on the left side the patient reacted. On March 31 the right arm showed violent clonic contractions. During April clonic contractions of the right side were frequently noticed, the left side being much less involved; plantar flexion on the left, extension on the right.

He became progressively feebler, and died at 1 P. M. on May 7, 1907.

CASE 6. S. G., 44, driver; admitted to Manhattan State Hospital August 15, 1906.

*Family History.* Negative so far as known; the information was very meagre.

*Personal History.* The patient was born in Russia in 1862; according to his own account he developed normally, became a cab-driver, married in order to please his mother, but separated after one week. His habits were irregular; he admitted gonorrheal infection, but denied syphilis. He married for the second time in 1894, had four children, one of whom died of "summer complaint." His wife had a miscarriage two months after marriage. The patient came to America in 1892 and worked steadily as a driver in New York City, being temperate in the use of alcohol according to his wife. Almost as far back as 1896 the patient had attacks of pain, chiefly in the left leg; the pain would come on suddenly like a shock and would jump from one place to another like "pins and needles;" occasionally the pain would go into the right leg. In 1900 he began to complain of a numb feeling in the left foot. On one occasion he was brought home in a wagon owing to weakness of the left leg; at that time he complained of pain in the calf and foot; his wife noticed no weakness of the left arm nor face, and there was no speech nor swallowing difficulty. He was in bed for five weeks. He appeared to recover completely, walked without any limp, resumed his work.

After this left-sided attack he was more inefficient at work. In 1903 or 1904, he fell off his wagon and sustained a fracture of the left arm; one finger had to be amputated. He was now cranky and irritable, his gait was ataxic; he was sent to Nebraska by the Hebrew Society. His wife tried to carry on a boarding house there, but was unsuccessful owing to the disagreeable behavior of the patient. They returned to New York and he peddled fruit and pretzels; he imagined that he was making his living, but in reality lost money. He now walked like a drunken man. He was considered to be insane in 1905.

In October, 1905, he was admitted to Montefiore Home; after a few months he began to be very expansive; in June, 1906, he was sent to Bellevue Hospital, where he boasted of his magnificent voice, of his great strength and weight, which he said was 205 lbs. (148 lbs.). He said that he had received $500 for singing. He was sent to Central Islip State Hospital and there showed the classical symptoms of general paralysis with a well marked tabes. He was able to sing for

the president, could earn $1,000 a week, a princess was in love with him when a boy, he could burn a woman with his look; his sister was worth $25,000,000.

After two months he was transferred to Manhattan State Hospital.

*On admission* to Manhattan State Hospital (August 5, 1906,) the patient was quiet and agreeable, smiled pleasantly and co-operated well in an examination; he answered questions relevantly, and the somewhat fragmentary nature of his utterances was probably due to his difficulty with the language. He was very much elated, boasted of his vocal powers and his great strength, but said that he had not one cent. He said that he saw his dead child at night and that he heard people singing at night time. He gave the date as February 30, the place as Bellevue Hospital, 26th street. He gave correctly the main facts of his life, but was not accurate as to the age of his children. He had a rather defective memory for the recent past, although he could give a general account of having passed through three hospitals. He showed no very marked defect in his grasp of general information. He counted 38 cents correctly and quickly. He had absolutely no insight into his physical and mental impairment.

*Physical Condition.* A very well nourished man, with no external evidence of syphilis. Slight ptosis of the left eyelid; pupils unequal, reacted on accommodation, but very sluggishly to light. Absence of knee-jerks and Achilles-jerks; plantar flexion on the two sides. Marked ataxia; tremor of fingers, tongue and facial muscles. Speech tremulous and sticking with distortion of words. Writing untidy, his name being almost illegible and showing marked tremor. General diminution of pain sense.

During the summer the patient continued to be quiet and good-natured; he gave the year as 1606 or 1506, and then again as 606. He still maintained that it was Bellevue Hospital and denied that there were any crazy people about. He did not give any relevant answer when questioned about hallucinations.

*On September 17*, he fell and sustained a fracture of the *left* thigh, which did not unite. The fracture became compound and in December the end of the upper fragment was sawed off without there being any necessity for an anesthetic; the skin was sutured and the wound healed well. He continued to have grandiose ideas and talked of his relations to prominent princesses and actresses, his ideas had a marked sexual trend. By May he was able to bear his weight on the injured leg, and to hobble about the room with support.

*On June 4, 1907*, he had a general convulsion lasting about half an hour, followed by irregular twitching for several hours. During the following week he had several convulsions; there was almost constant twitching of the left arm and occasionally of the left leg. *On June 6*, there was weakness of the left face and upper extremity; he had a considerable amount of movement of the left leg; touch and localization were impaired over the left face, arm, trunk, leg; there was left-

sided hemianopia. The pupils were unequal, showed no reaction to light, and defective reaction on accommodation. Ptosis of left eyelid. *On June 11*, the convulsions had ceased; he was unable to move the left side and had left-sided hemianopia. He said that he had been killed on the previous day and that he was now dead. He wished all the medical men to come and examine him.

After two weeks the left side was somewhat stronger. He was rather irritable and abusive.

During July there was no change in the patient's general condition. Although the left side was weak, he was able to get from his bed to the chair and back without assistance.

*On August 12*, the left arm and leg began to twitch, without involvement of the face; the contractions were more frequent than the heart-beats. There was definite impairment of sensibility on the left side. There was no impairment of consciousness; he was talkative, referred spontaneously to the twitching, said that he was certainly going to die in the evening, and that he would therefore like to go home to his wife and arrange about the insurance. He said that he did not feel pain, but was annoyed by the twitching; "I know I am dying, I want to speak with my three children, to be good to the mother when I am no more, I am very sorry when I must die, I will be 45 on Christmas, do for me what a poor man, a dying man, expects, put me in two blue blankets and in the ambulance, want to die with my woman." He accepted comforting assurances gratefully, and when told that he would be all right he said: "Really, well then all right; when you explain me that I wait." Next morning the left leg and arm were at rest, but on the left side in the region of the head and thorax there were irritative symptoms. The sternomastoid and scaleni were felt to contract strongly, the head was jerked to the left, the left side of the thorax was simultaneously affected, but the pectoral was not felt to contract. The diaphragm contracted synchronously with the above groups, the right side of the thorax did not participate in the frequent jerky upheavals of the left side.

*On August 14*, in the forenoon there was occasional twitching of the left leg, arm and muscles of the neck, but not of the face; in the afternoon no twitching was observed.

*August 29.* Since the last attack the left side has remained slightly weaker and the patient can no longer get to the chair without assistance. The left-sided hemianopia persists and there is diminution of the sensibility on the left side, except over the face where no difference between the two sides can be demonstrated. Slight left-sided ptosis. The fingers of the left hand tend to be flexed and stiff, but can be straightened out.

The patient was euphoric, but made no boastful statements. In giving an account of his life he was quite unable to correct glaring discrepancies.

*October 13.* Last night the patient had a brief general convulsion. This morning the patient has returned to his previous condition.

*November 4.* Twitching of the left face, leg and arm for one and one-half minutes, loss of consciousness for one minute; return to previous condition.

*November 6.* Last night and this morning left-sided convulsions, not carefully observed. The left arm this morning is no weaker than before; it frequently moves in a jerky fashion and he holds it with the other hand. He is able to move the left hand awkwardly. Left-sided hemianopia and diminished sensibility.

*November 17.* Last night the patient had a general convulsion which began on the left side. To-day he is in his usual condition. Two weeks later the patient began to have a series of general convulsions, in the intervals between which twitching of the left side persisted.

*November 30.* This morning he began to have twitching of the left leg and arm (face not mentioned); at noon he had a general convulsion, preceded by a short cry. He lost consciousness for about one minute. On regaining consciousness he appeared frightened, said that he was sick; he wanted an ambulance sent for. During the following night he had a general convulsion.

*December 1.* Slight twitching of the left side persists, but does not interfere with his voluntary movements. He seems happy, laughs and talks to himself.

*December 2.* During the night and this morning he had severe convulsions. Twitching of the left side persists but the jerks are quite far apart. The head is tilted to the left, and he can not turn it to the right further than the median line. The twitching involves the head and draws it to the right. The head, arm and leg jerk synchronously.

The patient is depressed, thinks that he will die soon; he can easily be induced to smile.

*December 3.* Convulsion last night. The twitching continues but is less pronounced and with longer intervals between the movements.

No further convulsive attack occurred at this time; the twitching continued during the following few days.

*On December 9*, it was noted that the weakness of the left side had become more pronounced after this series of convulsions; the patient could barely lift the left arm from the bed.

During the first half of 1908 the patient was not observed very closely; he was noted in March as having a convulsion, with special involvement of the left side; on May 1, he had twitching of the left side, which lasted for a few hours.

*November 12, 1908.* The patient's mental condition has changed little in the past year; he still is grandiose, talks of palaces and millions. Knee-jerks absent; plantar flexion on the right side, no plantar flexion on the left. Left hemianesthesia and hemianalgesia almost complete, less marked over the face. Left face paretic; considerable

weakness of left arm; he can not lift the left leg from the bed. The left eyelid occasionally droops a little but can be moved voluntarily as well as the right. Argyll Robertson pupils. Very pronounced lymphocytosis of the cerebro-spinal fluid; butyric acid test positive.

On later examination the plantar reflex on the left side was more of the extensor than of the flexor type; a response was difficult to elicit; there was an occasional extension, occasional flexion. Oppenheim—slight extension on the left side.

During 1909 the general condition of the patient showed slight further deterioration. The neurological symptoms remained the same. July 23, 1909: Argyll Robertson pupils, knee-jerks absent, ataxia and hypotonia. Flattening and weakness of the left side of the face, weakness of the left arm and leg, occasional extensor response of the big toe. Left-sided hemianesthesia and hemianalgesia; left hemianopia. Left-sided ptosis. Marked tremor of the tongue and fingers, speech slow and slurring with great distortion of test words. On September 23, it was noted that the fingers were flexed on the left hand, the hand on the wrist, and that they could be only extended to a slight degree. The mental condition showed little change; he gave his age as 37, 46, 36, 47; he had married three years ago at 15 or 16.

On November 5, 1909, in the evening the patient became unconscious and remained so until his death; the *right* side of the face, right arm and leg were twitching, and continued to twitch with intervals until death.

On November 6, 3.25 P. M. the patient died.

CASE 7. R. F., 34, actor, right-handed; admitted to Manhattan State Hospital April 13, 1905.

*Family History.* Negative.

*Personal History.* The patient was born in the United States in 1870, was a healthy, intelligent child; at 14 he became a compositor; at 20 he went on the stage; about 30 he married an actress, but separated after a few months. He himself admitted having had a chancre and skin-rash, but could give no details; he was addicted to the use of alcohol.

For several years before admission the patient had violent outbursts of passion; *in 1903* he had difficulty in getting work, probably because he had difficulty in learning his parts. In the summer of *1904*, when he came back from a tour, his speech was very defective and drawling. In *October* he visited a friend, was *unable to speak;* instead of going home he went to pay another visit, was found *unconscious* and taken to a hospital where he remained for six days. No further details of this apoplectiform attack could be obtained. On admission to the hospital he was diagnosed epilepsy, and on discharge acute alcoholism. Some time after this he had hallucinations one night, saw all sorts of animals on the ceiling. During the winter he remained at home, showed no desire to work. On *April 3, 1905*, he went out for

a walk, had a *wandering episode* and was heard of seven days later in Bellevue Hospital; he could give no account of his wanderings. There he was extremely stupid, made *almost no replies to simple questions*, answered "yes" when asked if he was strong and well; as a rule he merely smiled instead of answering. He was committed to Manhattan State Hospital on April 14, 1905.

On admission he was simple, childish and elated; he did not know where he was; he gave the month as March, and again as June. "I am in elegant health, I was never sick in my life, I am an athlete, I am an actor and I am as good as any of them in my line, nothing worries me, nothing bother me, I have no troubles and I am very happy." His memory was very poor.

*Physical Condition.* Knee-jerks exaggerated; pupils sluggish; speech defective; tremor of facial muscles; gait and upright position unsteady; lymphocytosis of the cerebro-spinal fluid.

During the ensuing year he showed little change. In *October, 1905*, he had several convulsions. He thought that he was in a hotel, could not give the date, had very poor memory both of the remote and recent past.

In *July, 1906*, he had transitory weakness of the *right* hand and the right side of the face; no sign of Babinski; the duration of the weakness was not noted. On *October 23* be became stupid at breakfast, did not take his food, was carried to bed. There he lay with his eyes open, but made no reaction to questions and demands. There was no asymmetry of the face, either when at rest or during yawning; the strength of the facial muscles could not be further tested. There was marked weakness of the *left* hand; he could not stand, made no effort to support himself; impairment of sensibility on the whole of the left side—face, arm, trunk and leg (defective reaction to pin-pricks *i. e.* slight uneasiness, and only occasionally well-directed movements); apparent *left-sided hemianopia* (no reaction to feinting from the left, no attention to objects in the left visual field); plantar flexion on both sides; marked exaggeration of all the deep reflexes, no difference between the two sides. He took dinner well and in the afternoon could make a few, almost inarticulate remarks. (How are you?) "All right—read—re—re." (Your name?) "Re—whe—whe—le—red—read." Next day he showed little change, but was able to swear freely. On *October 26* the motor and sensory disorder had disappeared, the apparent *left-sided hemianopia* persisted. He did not recognized his mother, said that she was dead; he occasionally laughed and talked about "bastards," "sons of b——," etc. On *October 28* he was seen in the forenoon swearing furiously with head and eyes directed *towards the left side* as if in response to visual hallucinations in the apparently hemianopic field. In the afternoon he was quiet, answered questions relevantly. On *October 31* he read print promptly but with some mistakes. *The hemianopic disorder persisted*, and as a rule the patient lay with head and eyes turned to

the right, and showed no interest in the world to the left. If pricked by somebody on the left side he swore at the man whom he saw on the right. His utterances were limited to fragmentary oaths; he lay in bed, gritting his teeth loudly, occasionally shouting peevishly and swearing, when not disturbed. He became steadily more emaciated, and much contractured, so that for the last three months of his life he lay folded up with thighs on abdomen, heels on buttocks. He used the *left* hand, which was the colder of the two, less than the right.

In *February, 1907* (examined February 9, 26), there was well marked *diminution of sensibility* on the whole of the *left* side, and he did not grasp at all with the *left* hand; the apparent left-sided hemianopia was permanent. The plantar reflex was flexion on both sides; the pupils showed very slight and sluggish reaction to the electric lamp.

On *March 13*, at 9.45 A. M., the head and eyes were turned to the left; the eyes were somewhat raised, moved about uneasily, never came to the right of the middle line. The right arm was rigid and flexed with the right hand at the throat, the fingers completely flexed. It could only be extended with considerable force, then gradually it returned to its previous position. The left arm was slightly stiff, the fingers only semiflexed. Plantar flexion on both sides. The patient was unconscious. On *March 14* the patient was in the same general condition, the eyes, however, occasionally came to the right. The *left* cheek was flatter than the right. On *March 15* the eyes were directed to the right, and the patient reacted promptly to feinting from the right side. On *March 30* the eyes were turned to the left; he showed no reaction to a lighted match even in the right visual field.

The patient had become steadily more torpid, but took his food eagerly up to the end; the excretion of urine was very variable, on two occasions within the last week he only passed two ounces in the 24 hours (removed by catheter).

He died in a state of profound emaciation on April 5, 1907.

CASE 8. A. S., 26, laborer; right-handed; admitted to Manhattan State Hospital January 22, 1906.

*Family History.* The patient's father was a heavy drinker. Nothing further was known of his antecedents.

*Personal History.* The patient was born in West Virginia in 1878, developed normally but received a meagre education, worked as a bootblack and later as an efficient riveter; he was very intemperate. About four years before admission to the hospital he had a chancre (brother's statement); nothing was known as to treatment. During the winter 1904–05, he became inefficient; "he would once in a while act simple, then pick up again." If asked a question he would ramble on irrelevantly; when told to do anything he would simply laugh and whistle and not do it. His speech became progressively worse during the six months previous to admission. From October to December,

1905, he worked cleaning dishes at a lunch counter for two or three hours a day. At no time previous to admission were ideas of greatness or other morbid ideas noticed by his brother.

In January, 1906, a few days before admission to Bellevue Hospital, the patient was sent to the laundry; three days later he was found in a hospital in New Rochelle, where he had been taken after being discovered in the woods with his arms round a tree. *He could not talk* to his brother when visited in the hospital, but would only laugh; he tried to talk but something seemed to catch his speech (the above anamnesis was obtained from his brother in May). He was transferred to Bellevue Hospital. There he showed a great difficulty of expression; "I feel—feel—feel all—all—all—all right—I am twenty—twenty—twenty two years" (26). The patient was apt to repeat the last few words of the question, and only answered after much urging and in a stammering way.

*January 22, 1906.* Admitted to Manhattan State Hospital. The patient had a dull, drowsy appearance; he had to be assisted in undressing and bathing himself. In bed he lay quietly, seemed to take a languid interest in the environment. He took his food well and tidily and was cleanly. He smiled to the physician but only answered questions after a preliminary pause and with considerable difficulty. He promptly gave his hand when asked to do so, but later showed some disinclination to be examined, withdrawing his legs and covering himself, without showing active antagonism. He occasionally tried to leave the room, but gave no reason for it; he could not be brought to kneel on a chair (to test the Achilles-jerk), insisted on standing up on the chair and holding his leg in various awkward positions.

His mood was one of mild good humor; no morbid trend was elicited. He gave his name when asked, said that his age was 20 (26). He had a general idea of the environment and of the season. (What kind of a place?) "This stol—pol—sstol—pol—isn't it spol?" He gave no spontaneous account of the past; his answers were fragmentary, elicited with difficulty and sometimes contradictory. He said that he was born in "W—W—jest—W—gi—g—W—W—W—Virginia" (correct). (What is your work?) "My work—rai—rail road—railroad." (Engine driver?) "Driving rivets." (Where did you work?) "Every G—d(?)—every railroad (points out of window)—every 9th Ave—6—6—3rd—3rd—3rd was the—that was all the railroad." He denied any memory of Bellevue Hospital, but had a correct grasp of the duration of his stay in the present hospital; he could give no account of how he came here. His grasp of general information showed extreme dilapidation; he was unable to do a simple multiplication; 4×5? "6". He did not grasp a test name nor number well enough to retain either, even for the shortest time. He had no insight into his condition, said that he was strong, that there was nothing wrong with him.

*Physical Status.* The patient was a very muscular young man 4 ft. 4 in. in height; no anatomical stigmata; nutrition satisfactory; evidence of recent frost-bite; old injury of thumb. There were several syphilitic scars on the shins; he admitted having had a chancre, could not give a trustworthy date. The pupils were slightly unequal (left smaller than the right) and showed the sign of Argyll Robertson. The left knee-jerk was active; the right knee-jerk was not elicited on the first examination, but on a later examination was found to be present but very feeble. No other Achilles-jerk could be elicited. Plantar reflexes; smart flexion on both sides. There was no local sensory disorder; he localized well on touch with a brush; there was general diminution of the sense of pain. There was no motor weakness. His gait was ungainly, but not ataxic; he was able to stand steadily with his feet together. There was marked tremor of the tongue and facial muscles, but none of the fingers. Speech was extremely defective with tremor, sticking, and marked distortion of the test words; *e. g.* (Methodist Episcopal), "kisc cop cop;" "mest-a-ist-ist-a-ist oc;" "mist-a dis-a dist-oc-al;" (third riding artillery brigade) "thir-till-ll-i-go;" "ah-till-ll-r-r-go." In attempting to write the patient merely produced a tremulous distortion of the initial letter of the word asked for; he could not write his name. Organic reflexes under control. Slight peripheral arteriosclerosis.

*Disorder of Speech.* The patient showed a disorder of speech which was more than a simple dysarthria. He understood simple commands, although he occasionally failed to co-operate. He answered simple questions as regards his name, age, birthplace, work, orientation, etc. His occasional failure to respond was attributed to the general mental reduction, and not to a specific disorder in understanding spoken speech. In expressing himself the patient spoke slowly and showed marked dysarthria; at times his utterances were inarticulate, and he would make use of gestures. He frequently repeated syllables, especially the first syllable of a word. (How do you feel?) "How—eh—eh—fee—feel—feel." (Are you sick?) "Si—si—pray—why." (Name?) "My na—na—na—is Smith." (Correct.) At times he used paraphasic utterances. (Are you sick?) He laughs, much amused, "Sick—turk—turk—turks—scru—scru—crose (? clothes)—s-s-s—and a bum, a bum—see—der—Andre (his brother Andrew)—bu—butt—button (? referring to shirt)—they're on-da on-da on-da bum."

The patient gradually became more alert; his speech improved, but he was still exceedingly difficult to understand owing to his stammering repetitions and general difficulty of expression. From time to time during the first week in the hospital he got out of bed, hunted under the bed for fairies. He was rather dull, but occasionally picked up the remarks of other patients and laughed at them. The following is an example of his utterances (January 30): (How did you get your finger hurt?) "I got that—to—taken off—wor—work—

machine—with a machine—bottle—was put—putt—putt n—a co—cork in see and that see it—co can—went in right there see."

By April the patient had become an active and willing worker in the ward; his speech still showed marked stammering and sticking; he would frequently give up the attempt to express himself and smile vacantly. (What place is this?) "Sta—sta—state." (Q. repeated)—"Jus—jus." (What for?) "Jus four yar—yo—jes—jes". In May he was able to express himself better; he gave fragmentary information about his past; his memory was very poor and confused. During the summer he remained at the same level.

*October 2, 1906. Apoplectiform attack: right-sided weakness of arm and face and hemianesthesia; apparent right-sided hemianopia, twitching of right face and platysma.*

Before dinner the patient was unable to carry in the tray, which he had usually carried in. At dinner he became pale, his head fell forward; he was placed on the floor, a large piece of tomato was removed from his throat. He was placed in bed. He there made inarticulate sounds, appeared to be trying to talk, he was distressed, made continual restless movements. *He made no response to written or spoken demands;* he gave an equally feeble handgrip on the two sides. In the afternoon he became somewhat quieter. At 6 P. M. the *right side of lower face and platysma* began to twitch simultaneously; he began to show continual restless fumbling movements in which he used only the left hand; he seemed unable to grasp the clothes with the *right hand*, he fumbled the right hand with the left as if the right were numb or a foreign body; he gave a weak hand-grip with the left hand, *no grasp with the right. He was able to stand up without support.* There was no sign of Babinski. His reactions to pin-pricks on the two sides of the face were equally variable; he did not react to pin-pricks over *right* arm and leg, reacted sluggishly on the left side. The eyes never came to the right of the middle line; he could not be induced to look to the right; feinting at the eyes showed that there was an apparent *right-sided hemianopia.* The patient soiled the bed; he required to be catheterized. The urine showed no albumin.

*October 3.* Twitching of *right platysma* and of *right side of face;* occasional twitching of *elevator of right ear.* The right side of the face was weak. The patient became brighter in the evening, but articulation was still poor although one or two words were intelligible.

*October 4. The right side of face, right ear and platysma* still continued to twitch; right face was weaker than left; the patient began to move spontaneously the right arm much more. He showed generally diminished sensibility with no difference between the two sides. Feinting at the eyes demonstrated the same apparent *right-sided hemianopia;* but the patient from time to time spontaneously moved his eyeballs to the right. He was generally brighter, but practically inarticulate; he was uncleanly.

*October 5.* Right side of face continues to twitch, but not the platysma nor ear. The patient is generally brighter.

*October 6.* Twitching has ceased. The mouth is pulled to the left side; the right side of face moves much less than the left. The patient is, as a rule, inarticulate, but twice said "yes." He looks bright but does not carry out spoken nor written orders; when given a magazine with the page upside down he reverses it. He makes no effort to pick out objects named, shows no objection when wrong names are given to them. Sensibility to pin-pricks over the right arm seems impaired, but he reacts to pin-pricks over the fingers. Over both legs the reactions to pin-pricks are the same. He does not react to feinting at the eye from the right side so constantly as from the left.

*On October 7* there was some twitching of the right side of the face which stopped three hours after pot. brom. gr. 30 and chlor. hydr. gr. 20 were given.

The patient was allowed up on October 9; he was still unable to speak; the weakness of the right face persisted but no gross weakness of the right hand was present. It was not noted whether the sensory defect and the hemianopia persisted.

*On October 12* twitching of the right side of the *face* reappeared without any loss of consciousness, while the left side of the face showed some coarse tremulous movements, but not the definite clonic contractions of the right side. No weakness of hand could be demonstrated; the right side of the face seemed less weak than on October 9. Although articulation was extremely poor, the patient had in the last three days made so much progress that he was partly intelligible. He spontaneoasly gave a few fragmentary reminiscences of his life before admission, referred to his brother's liquor store, to his own work on the railroad and dock. "I'd like—I'd like—go home—I con—num—num—10 W. Street—W. Street—down (points)—liquo—go—liquor store—number 10 West Street—it's liquor do Andrie—no ga—ga—that's my brotha—n—n—Jack—that's—that's—Jack—that's—the—the partner—with—wi—he's—hotel—eh—ah—I was—I was pa—partner (grins)—I was under Ado—I—I worked on a rados—id—wa—on train—you know—ah—I ten—ten years worked on—ah—on railroads—that right—I have—ah—ah—I don know—I don know—ah—*pah blaws—arn yous*—was on the dock—*oranges*—."

The disorder was not merely an articulatory one, but to a certain extent seemed to depend upon a special difficulty in the higher speech mechanism.

At first the patient did not respond to spoken questions or commands, but after some time he gave his name correctly, his age as 60, 40; he showed considerable perseveration. He picked out a knife when it was named, read a series of numbers on a tape-measure, made confused attempts to add up small change. When asked to write he made an initial scrawl, dropped the pencil, said "I can't."

For the next two weeks the patient went about the ward, talked a

little, occasionally showed twitching in the right face, which was especially well marked when he smiled.

*On October 28*, he appeared dull, confused and flushed; he was put to bed. *The right side of the face was occasionally twitching;* it was not possible to test the strength of the limbs; the patient did not react to pin-pricks on either arm. He made peculiar clucking sounds with his tongue, which lay in the left side of his mouth. He laughed, "a-ha-ha." Your name? "Ant-o-ty," (Anthony) said with tremor, especially of the right face, and only after one or two efforts. He made no endeavor to name objects. The test for hemianopia was inconclusive. Knee-jerks: right not elicited, left brisk. Wrist and triceps reflexes equally exaggerated on the two sides. Plantar flexion on both sides.

During the next month the patient was continually restless and fumbling with his clothes, grinned foolishly when interviewed, began to talk a little.

By the end of November he answered a few questions, talked in a *high-pitched*, tremulous, *almost inarticulate* voice; he frequently was unable to articulate but made *high-pitched, squeaky sounds.* He gave his name; continued to repeat his name in answer to several other questions. Are you sick? "Sick—no—no—I aint sick." Why are you in bed? "Ant—y Smith." (Q. repeated) "I don't know—wha—wha—." His answers to simple orders were extremely defective. (Give me your hand!) He holds up both hands. (Close your eyes!) No reaction. (Show me your tongue!) He does so. There was no definite weakness of the limbs of either side; the hand-grips were equal; he walked as if dazed, but with no weakness of either leg and no definite ataxia. The right side of the face moved less than the left (December 3). It was not possible to make any conclusive test for hemianopia.

In the beginning of December, the patient had a febrile attack, during which the heart sounds became impure, but no definite symptoms of pericarditis nor endocarditis were observed. On December 18, he recognized a visitor, the owner of the coffee-stand where he used to work.

The patient continued in the same general condition until the middle of January, 1907; he was quiet as a rule, but occasionally resisted while being cared for; he had no control of his reflexes. *He was quite unable to speak.*

*On January 16*, it was noticed that the left side of the face was continually twitching; from time to time there was a definite jerk of the left shoulder and left arm. While the left arm showed definite clonic movements, the right arm showed constant tremulous movements; *the right arm was continually occupied as if in carrying food to the mouth.* He continually made movements of the mouth as if to grasp something with his lips; he licked the tips of his right fingers. Pin-pricks over the left forehead caused vague distress.

*On January 17*, the irritative phenomena were most marked on the *right* side; both legs remained unaffected. The *right* arm and first two fingers of the right hand were twitching, 102 to the minute, as if a series of electric shocks were being sent through the muscles. The left arm did not show the same distinct twitches, but moved about restlessly and showed a tremor of wide range. Both sides of the face and the forehead were twitching; the head and eyes were turned to the extreme *left*, the eyes being also directed upwards.

*On January 18*, the left arm continued to show the same tremulous, jerky uneasy movements; the right arm remained quiet, but showed occasional jerks; the right face twitched vigorously, also the frontalis. The eyes were still directed upwards and *to the left*. No definite reaction to pin-pricks or pinches anywhere could be obtained. Plantar flexion on both sides.

The patient frowned when interfered with; when shouted at he brought his eyes into the middle line. He was able to swallow liquids with some difficulty.

*On January 19*, the movements showed the same character. The left arm was tremulous and restless; the right arm was quiet with an occasional decided twitch. All the muscles of the face were twitching, but the right side of the face more so than the left; the lower half of the left side of the face twitched less often than the upper half; the ears twitched from time to time. He frequently sucked his left fingers. The eyes were now turned to the right.

During the next few days the movements continued to show in general the same character and local distribution.

*On January 21*, there was slight left-sided ptosis. Tests of the visual field—feinting at the eye, and moving a lighted match—pointed to *left-sided hemianopia*. In the facial movements pursing and sucking movements of the mouth were very prominent. The *left leg* seemed to show more impaired sensibility than the right, but apart from this no difference on the two sides was noticed; he reacted by symptoms of distress to pricks over the face, shoulder and arm on either side.

*On January 24*, the patient had practically ceased to show any twitching and appeared much brighter. During the past weeks he had emaciated greatly.

*On January 25*, at 9.30 A.M. the eyes suddenly moved to the extreme left, moved uneasily; the eyelids were half closed; the mouth was pulled to the *left* and showed sucking movements, the fingers of the left hand fumbled the lips in a tremulous manner. The patient continued to show frequent twitching of the face, most marked on the *left side* with exacerbations in which the face was pulled to the right. No accurate test for hemianopia was possible, but while he winked when feinted at from the right, there was no such definite reaction when feinted at from the *left*. In general he reacted vaguely to pin-pricks, but made no reaction to pin-pricks over the *right* arm. He was able to walk with support. Plantar flexion on both sides.

During the following days the twitching of the facial muscles, restlessness of left arm, with comparative immobility of the right arm, continued.

*On January 26*, slight ptosis of the left eye was noticed. No reaction to pin-pricks was obtained over arms, legs, left forehead; he winced when pricked over right forehead.

*On January 30*, he frequently made restless grimaces with both eyes closed; he frequently sucked his fingers, occasionally gave a high-pitched cry.

During the next week the patient's condition was specially characterized by the facial contortions; his face was frequently twisted into a variety of strange involuntary grimaces. He occasionally uttered a high-pitched cry especially when disturbed.

*On February 3*, there appeared to be no hemianopia; he winked when feinted at from either side.

*On February 7*, his tongue seemed unable to move the food back in the mouth; he was accordingly limited to liquid diet.

*On February 10*, the patient showed marked convulsive grimacing, frequent yelling in a high-pitched voice, with mouth wide open, tremor and jerky movements of the *left* arm, uneasy movements of the right arm. The eyelids would close tightly, the left upper being pressed down over the lower eyelid. No satisfactory sensory examination was possible. Any disturbance of the patient provoked general facial contractions. Plantar reflex was flexion on both sides.

*On February 18*, he winked when feinted at from either side. On February 26, apparent left-sided hemianopia was observed, and this was still present on March 1. He paid good attention to objects in the right visual field, none to objects in the *left;* he only winked when feinted at from the right side.

*On March 7*, the patient was shouting loudly, grimacing vigorously; there was an indication of right-sided hemianopia; he winked when feinted at from the left, not from the *right;* the eyes were turned to the left, did not come to the right of the middle line. He licked the fingers of his right hand. He fumbled about equally with the two arms. He reacted to pin-pricks all over the body. He frequently yelled, the yell was a sudden loud shout ending frequently in a series of muttering sounds or in a series of choking sounds; there was frequently a tremor in the shout, and sometimes a stuttering interruption and violent movements of the tongue and lips as if for articulation.

*On March 16*, the patient showed definite evidence of left-sided hemianopia, which persisted until his death on *March 25;* he followed promptly objects on the right side, but did not at all react to objects in the *left* visual field. He showed frequent grimacing, the tongue was in constant motion. During the last weeks of his illness he showed extreme emaciation and progressive contracture of the lower extremities.

He died on March 25, 1907.

CASE 9. J. D., 35, right-handed; separated from second husband; tobacco worker; admitted to Manhattan State Hospital, February 11, 1909.

*Family History.* The maternal grandfather died from apoplexy at 71; the maternal grandmother died at 71, having been bedridden with paralysis for twelve years. A maternal uncle died from apoplexy at 62. The father died from apoplexy at 39.

*Personal History.* The patient, a right-handed woman, was born in 1874, she developed normally, received little education, and went out to work at an early age. At 23 she married; four months after marriage she had a miscarriage, then a still-born child at seven months, later a live child who died at seven from cerebrospinal meningitis. Her husband died in 1903, having been bedridden for over two years. There was no history of syphilitic infection, nor of secondaries. In 1906 the patient remarried, but after a year was deserted by her husband and went back to work. Before the stroke in July she had already appeared somewhat nervous and her hands would shake.

On July 17, 1908, after getting out of bed the patient fell, but did not lose consciousness; she could not move the left arm, nor leg, the face was twisted, the speech was thick and difficult to understand. She was taken to a hospital. The left leg and arm were stiff and paralyzed; the tongue deviated to the left, there was no sensory disorder. The reflexes of the left side were exaggerated; no sign of Babinski nor ankle clonus. The eyes could not be moved to the left, but the movements were free in all other directions (? left-sided hemianopia). At times her talk was delirious. Her speech improved within a week; in two or three weeks she began to move her hand and arm and in another week could move her leg. She complained of a feeling of heaviness and numbness in the left side. She required to be catheterized. Her sister noticed that the patient showed some difficulty in understanding things; she was correctly oriented. After seven weeks she left the hospital, walking with a typical hemiplegic gait. During the following four months the patient showed progressive deterioration; she would sit and talk to herself, her memory for the recent past was poor, she would put things away and forget where she had put them. She occasionally fell without losing consciousness; she complained of numbness and pains in the legs (? leg). In the middle of January, 1909, she was taken to a hospital, where she was frightened by the colored nurses; two days after admission, she seemed dazed, did not recognize her brother-in-law. She was said to have been delirious and to have talked about having trunkfuls of diamonds. She was transferred to Bellevue Hospital as she had been screaming, yelling and uncovering herself. In Bellevue she was restless and confused, disoriented and incoherent; she talked in a paraphasic manner.

*On admission* to Manhattan State Hospital (February 11, 1909) she cried and resisted, behaved like a child, wept and protested when

brought to the examination room, was apprehensive and only reassured with difficulty. At times she became indignant, but could hardly express herself, owing to her defective articulation, complicated by a difficulty in finding the correct words. She was a little amused when her sensibility was tested. She answered some questions relevantly but showed marked perseveration and had frequently great difficulty in finding words. What is your name? "You told me, didn't I? My name is Josie, Josephine (correct). I did't tell you my last name" (articulated with great difficulty). Tell me about your trouble. "I'll tell you my last name; I'll tell you if you want to know—2, 3, g, do, 32 (35), that's true; I aint telling no lies; my last name is 2; my name 1, 2, my last name is fifty-five. Didn't I mistake my last name, 35. I tell you my first name, didn't I, didn't I tell you, I told you, well! have you got it? (turning to stenographer); my last name 35, I told you."

The patient at times was much irritated over her speech difficulty and, during the whole examination, gave no evidence of elation; she said, however, "I am always happy." She uttered no grandiose ideas; no delusions of any kind were elicited. She knew that she was in a hospital, although she could not find the word, "I can't think of it for a minute—of a sick people go in it, (hospital?) I can't think of it for a moment, all the sick people go into it, what is it? (she becomes irritated, the physician says hospital; she takes this up eagerly) that's it hosp, hosp, kospital, kospit-able—I belong downstairs and I came up to see you." When asked for the month she said, "this is third." Her memory was impaired, but her actual grasp of the past could not be accurately determined owing to the aphasic disorder which caused her to give absurd answers. She was conscious of the difficulty which she had in giving the facts—"I can't think, you know; my mind is a little wind, wander, you tell me, tell me questions."

Where were you born? "Was I? I must have been 244 at 44, wasn't I the same? I was born and then I suppose when I was born; I don't remember when I was born." She had difficulty in recalling the trip from Bellevue Hospital, but showed by gestures and eager acceptance of hints, that she had some memory of the trip. She had considerable difficulty in retaining a name for ten minutes. She admitted that her memory was impaired, recognized her difficulty in speaking and writing; "my mind is all right, yes it is; but if I was better, if I was better you know, I'ld be all right."

*Physical Status* (The patient was very excitable and irritable during the examination); a well built woman with residuals of a left-sided hemiplegia, weakness of the left arm and leg, sign of Babinski on the left side; the right side of the face appeared to be a little flatter and to move less than the left on admission; on a later examination the left side of the face moved slightly less than the right. The knee-jerks were much exaggerated on both sides; the gait was ataxic as

well as hemiplegic. An accurate test of the patient's sensibility was not possible; she was able to distinguish the point from the head of a pin over the face and hands and she reacted to pin-pricks over the legs. The pupils reacted poorly to light but better on accommodation. Her speech and writing were extremely tremulous and sticking. She was unable to write her name correctly and showed marked paragraphia. The patient had imperfect control of her organic reflexes; she sometimes asked to be raised but only when it was too late.

*Aphasic Condition.* The patient usually spoke with considerable effort; some phrases were pronounced quite distinctly and without any tremor. As a rule, she had great difficulty in finding the correct words when talking spontaneously. She would become tearful and irritated owing to this difficulty. She was able to name some objects but had difficulty with others and showed marked perseveration. She was able to repeat one line of poetry correctly but, when given another line to repeat, she uttered a mixture of all the names for objects which she had previously been asked for. She recognized the names of objects and carried out correctly simple spoken orders, such as, "give me your right hand."

She was unable to carry out a command if a trifle complicated.

"Put your left hand on your right ear." She said, "your right hand you told me, I put it on the left hand didn't I, my left hand on my right hand—what did you say ? left my arm my left side." She was unable to carry out the order. She would read some words well, but would become hopelessly confused over others, even though very simple. She immediately read correctly "hand" and "horse." When shown the printed word "pig", she said "that is p backwards there now, take it." She was able to spell "cat" correctly but spelt "dog" d-a-c. She was unable to write letters named. In writing her name she began it correctly, repeated one syllable and then wrote something which was not at all like her name. In tests for apraxia the patient was clumsy but showed no clean-cut apraxia.

During February and March the patient appeared to become slightly more unsteady, but otherwise showed little change. She was much more difficult to examine than on the day of admission and would become violently irritated if an attempt was made to look at her pupil; she would frequently get into a state of great confusion and irritation over her hair and the bedclothes. On one occasion she asked anxiously and tearfully "Where is my neck? I can't find it on one side or on the other." When her hand was put on her neck she excitedly said "No, no, no." After groping about for a minute or two, she suddenly laid hold of her ears and gleefully exclaimed "That's all right, I have my neck now."

The patient was observant of what passed in the ward, called the nurse's attention to little incidents. She usually called other female patients "he" or "she" and addressed the physician as "ma'am."

Treatment by mercury inunctions and increasing doses of potassium iodide caused no change in her condition.

On April 15, in the morning she pushed away the drink which nurse offered to her and appeared to be dazed. During the morning she kept picking up the clothes with her left hand and occasionally put up her left hand to her face. When examined at 10.25 A.M., she lay with head and eyes turned to the left, the forehead was twitching on both sides, the right ear was twitching, not the left; she made inarticulate sounds, during her attempts at articulation the left face moved less than the right. The right arm was limp; she moved the left arm freely up to her face. While being examined she began to vomit without much retching; her pupils showed no reaction to light.

At 10.45 A. M. the right forehead and ear were twitching, the mouth was pulled to the left. She appeared to smile during the examination; the right side of the face moved less than the left. There was no reaction to pin-pricks on the right side of the face, on the right hand nor on the right leg. She cried when the left face was pricked, promptly withdrew the left hand when it was pricked, made defensive movements when the left thigh was pricked but did not react to numerous pin-pricks over the left leg. The right leg was twitching, the foot jerking rhythmically about ninety-six times per minute. The plantar reflex was extension on the left side, could not be determined on the right side.

At 11.15 A. M. the head and eyes were turned to the left, the eyes never came to the right of the middle line. The patient did not react to feinting at the eyes from the right side, but reacted when feinted at from the left.

At 11.35 A.M. the right arm and right leg were twitching, the right face was at rest, the head was turned to the right.

At 3 P. M. the patient was lying quietly on her back with her head and eyes turned to the left. She moved her left hand restlessly to and fro. An attempt at ophthalmoscopic examination was vigorously resented and she used both her arms freely.

On April 16 the patient was much more alert; she was very irritable and for long periods would shout out "Leave it, leave it." In fumbling with the clothes she used her left hand freely. Her head was turned to the left as a rule, but she occasionally moved it to the right and seemed to take interest in objects in the right visual field (on the previous day she did not react to feinting from the right).

On April 17 she was very irritable but at times laughed and chuckled and no weakness of either side of the face was observed. She vomited her breakfast without any retching.

On April 30 she vomited in the morning and, when interviewed later, she was extremely dull. During May the patient became progressively weaker.

May 20. For several days the patient had had her head turned to the right side and talked as if addressing imaginary persons. At this

date she showed what were apparently definite reactions to hallucinations in the right visual field. She paid no attention to a hand held in the left field, but saw a hand held in the right field. During the following day the patient lay with her head slightly retracted and her eyes directed to the right. She looked about in the right field as if in answer to stimuli. She paid no attention to any objects shown in the left field. In the afternoon her arms and legs twitched for a short time. There was no difference of reaction to pin-pricks on the two sides. She reacted promptly when feinted at from the right, did not react when feinted at from the left. During the following week the patient showed practically the same condition. She appeared to react to hallucinations in the right visual field. She wood look excited and say "I see them, I see them," and would stretch out her hand, left or right, as if towards some interesting object in the right visual field. When pricked on the left hand by the physician on her left side, she would indignantly tell the other physician on her right side to go away. During June and July, the apparent left-sided hemianopia continued and the patient only paid attention to what happened in her right visual field and appeared to react to hallucinations in that field. Any attempt at physical examination was violently resented by the patient.

The patient was restless, irritable, screamed loudly whenever cared for, required to be spoon-fed, took her food well. It was never possible to do lumbar puncture.

In the second week of August, a deep bed-sore developed. She became progressively feebler, broncho-pneumonia developed and she died on August 23, 1909.

CASE 10. G. W., 41; admitted to Manhattan State Hospital, April 24, 1908.

*Family History.* Negative; information was meagre.

*Personal History.* The patient was born in Germany in 1869, received a good education, graduated from the gymnasium; he later became a cigar-maker, but for several years worked as a ship-steward. He came to America in 1891. He married at 24 and had two healthy children; he was divorced, then remarried. He was temperate in his habits; previous to admission he had stated that he had a hard chancre in 1892.

*In April, 1905*, the patient began to have some trouble in rolling cigars with his right hand, as his right arm would shake so much; owing to this difficulty he was unable to support himself.

*In February, 1907*, he fell down some stone steps and struck his head: "He was unconscious and delirious and lost his speech for three days" (report from the hospital). He was discharged after two weeks in the hospital, the diagnosis was "cerebral syphilis, general paralysis;" "left pupil larger than the right, no reaction to light, slow on accommodation; no nystagmus, no facial palsy. Slight

tremor of the right hand, none of the left. Deep reflexes active, no bladder or rectal trouble, no Romberg."

On his return home his wife noticed that he could not write with his right hand nor could he use his knife. She noticed no trouble with his right leg. His speech was thick; his memory did not seem to be as good as formerly. He now began to suspect his wife of infidelity. He was restless and sleepless, would talk at random, blamed his wife for his sickness.

He was readmitted to the hospital on March 31, 1908, complaining of headache of long duration, diplopia, weakness of the legs, trouble with his water. He was not able to give a good account of his symptoms, but said that he had first noticed twitching of the right arm when he attempted to use it, and later twitching of the right leg. He said that for two years he had considerable weakness of both legs. The right arm and the right leg showed slight weakness. On movement of the right arm and fingers very marked tremor was observed, but no sensory disorder was demonstrated. Slight nystagmus; slight left-sided ptosis; slight weakness of the left-side of the face; the patient complained of ringing in the left ear. The pupils reacted defectively to light and on accommodation, the left was the larger and reacted less than the right. Optic discs clear.

During his short stay in the hospital the patient had attacks of vomiting; on one occasion he had to be catheterized. The patient was discharged after three weeks in the hospital, four days previous to his admission to Manhattan State Hospital.

*On admission*, April 24, 1908, the patient was dull but adapted himself to the routine of the hospital; he was accessible to examination, became somewhat tired after an hour. He had a fair idea of the environment, gave the date as May, 1908. On a later examination he gave the day and the month correctly. He was somewhat depressed, complained of the attitude of his wife toward him, stated that she was his common-law wife (she was able to show a marriage certificate). He talked in a vague way of seeing and hearing things in dreams, but otherwise no evidence of hallucinations was elicited. He made confused and contradictory statements as to the ages of his children; at one time he said that his mother was dead, again that she was alive. He made many other contradictory statements, but was able to give a fair outline of his life and of the various incidents of the psychosis. He admitted having had a chancre when over 20. He had a fair memory of the recent past, showed somewhat defective retention of tests. His grasp of general information was quite satisfactory. He made some mistakes in simple counting. He had insight into his physical disorder and referred to himself as being a little weak in the head.

*Physical Status.* A well built and well nourished man; slight weakness of the left side of the lower face; no weakness of the occipito-frontalis; slight drooping of the left eyelid; slight weakness

of the right hand-grip; slight weakness of the right leg; cutaneous sensibility unimpaired. The eyes moved freely in all directions; there was some nystagmus present. The pupils were unequal, the right reacted sluggishly to light, well on accommodation; the left did not react to light at all and reacted slightly on accommodation. The knee-jerk was slightly more active on the right side than on the left; on the other hand the deep reflexes of the left arm were more active than those of the right. Plantar reflex; flexion on both sides. Coarse tremor of the tongue. When the patient grasped an object with the right hand, a very coarse tremor of the limb ensued. He showed a marked intention tremor in taking hold of a glass of water with the right hand. There was a marked coarse tremor on movements of the right leg. Slight tremor of the left hand and fingers, none of the left leg. His speech was stumbling and ataxic and was much more defective when under the influence of emotion. Marked lymphocytosis of the cerebrospinal fluid.

*On June 7* the patient had an epileptiform convulsion.

During the summer there was practically no change in the patient's condition.

*On November 17*, the patient had a general convulsion lasting fifteen minutes. During the following day he was extremely dull and quite irresponsive.

*On November 20*, the patient had not yet recovered his previous level. He appeared to be extremely stupid, could only utter inarticulate sounds, did not appear to understand some questions; he could not name objects but appeared to recognize the names when given to him. The convulsion had not left any motor residual.

*On November 23*, he was able to say a few words and name some objects, and during the following week his speech improved considerably. It was, however, more ataxic than before the convulsion.

*On January 29, 1909*, epileptiform convulsion followed by a short period of confusion. In March the physical condition of the patient began to become much worse. By the end of April he was unable to walk without assistance. His speech was at times very difficult to understand.

*On May 7* he had another convulsion. After June he was confined to bed owing to his ataxic gait.

The general neurological status (July 4, 1909) was practically the same as on admission. At this period the patient was talkative and decidedly euphoric; he said that he was happy and strong, that his head was quite strong; he denied that he was ever suspicious of his wife. He was correctly oriented, gave an outline of his life without definite dates. His memory of the onset of the nervous symptoms was inaccurate; his retention of tests was very poor; no insight into his deteriorated condition.

*On August 7*, the patient complained of stiffness of the right hand, and immediately after this had a general convulsion of five minutes'

duration. For the following two days the patient continued to have twitchings, which were more continuous on the right side than on the left; he did not recover consciousness; died at 12.45 A.M., August 9.

CASE 11. M. T., 38, laborer, right-handed; admitted to Manhattan State Hospital, June 24, 1907.

*Family History.* A sister was said to be peculiar; otherwise negative (information very meagre).

*Personal History.* The patient was born in England in 1868, was a healthy child, developed normally, received a simple education. After leaving school he worked with a brass-molder at first, then as a miner, a longshoreman, a bartender. No history of syphilitic infection could be obtained, and there was no external evidence of it. He married in 1890, and came to America about 1893, where he worked as a bricklayer's laborer. His wife had first a still-born child, then two children who died at three months, six weeks respectively, then five healthy children with one miscarriage due to an accident.

The patient had occasionally indulged in alcohol to excess, but for the three years previous to admission he had been quite temperate; he had been a healthy man and his wife knew of no symptoms previous to December, 1906. During Christmas week he had a bad cough, and on Sunday he suddenly felt a severe pain in the forehead as if he had received a blow; he went to bed and the doctor diagnosed "grippe." He had been working vigorously the day before. On December 31, at night, he imagined that he saw beautiful processions, but did not hear any voices; he was quite clear in his grasp of the environment. For a night or two he had similar visual hallucinations, but none during the daytime. He remained at home for several weeks (duration uncertain) feeling out of sorts but was not completely confined to bed; he then went to look for work and after some difficulty he got a job.

In April, one day, the left leg became weak, the left arm began to twitch, the face remained straight (wife's account); there was no speech nor swallowing difficulty. He walked home dragging his left leg, the left hand continued to twitch for several days, and he complained of a warm feeling in the left leg. When the twitching stopped there was no residual weakness; he went to work, and was quite efficient (according to his wife) but several times while working the left hand would begin to twitch and he would feel weak all over; plunging the hand into cold water seemed to stop the twitching, according to the patient. These attacks of twitching left no residual weakness. He told his wife that when he had the twitching little red lights would come in front of his eyes. On June 17, after working six hours, the left-sided twitching came on, he felt very weak and went to bed; the left hand continued to be weak and to twitch so that he went to Bellevue Hospital on June 19; when visited on the 23d, the twitching had stopped, and he appeared to be in his usual condition, according to his wife's statement.

He was committed to Manhattan State Hospital, as the physician at Bellevue found him "excitable and emotional, readily confused and perplexed; he declares that he sees shadows and peculiar motions of objects which he knows to be still; he shows some memory defect for recent events. His left arm and hand show some loss of power."

*On admission to Manhattan State Hospital,* June 24, 1907, the patient was very agreeable when interviewed, he did whatever he was asked to do, he accepted the environment without comment, and seemed quite contented with the treatment he received. When examined he answered questions promptly and relevantly; showed no disorder in his conversation; he told all his symptoms and said they were due to an attack of grippe at Christmas time. He said that about four months later, after he had been working only two days, "I imagined by my eyes that I saw fire and that those fingers (left) went that way as if the nerves closed them together." He had no absurd ideas, but was abnormally cheerful and optimistic in view of the actual environment, the nature of which did not seem to impress him; he did not at all realize the serious meaning of his physical symptoms and talked of a good job, which he expected to get soon. He had neither delusions nor hallucinations, but gave a retrospective account of seeing fire during the attacks of weakness; this visual disorder was not present in the earlier attacks, but during the latter attacks he imagined he could see three or four little round blazes about an inch in size right in front of him. They seemed to be about a yard distant, they did not interfere with his sight; he could drive in a nail even with them in front of his eyes; this phenomenon would last about an hour. He could only specify two attacks in which this phenomenon was present. One night in June, shortly before admission to Bellevue, when he went to the physician's house, all the people in the waiting-room seemed colored (they were really white). In Bellevue he had imagined that he saw colored persons coming to him, but when they came up to him the "reflection" cleared away and he saw them white. He gave the date correctly, knew exactly where he was. He had a fair memory of the main events of his life, but became confused and contradictory in his dates, showed glaring discrepancies which he could not correct even when they were pointed out to him; he married in September, 1890, came to America in May, 1892, his wife had three children and one miscarriage before he left. He had a good memory of the events preceding admission to the hospital, but did not remember having had a conversation with the examiner three hours previously. He retained the various tests well. His store of general information was satisfactory.

He had no insight into his memory defect, and denied that there was any mental impairment; he did not realize the seriousness of the rather striking neurological episodes.

*Physical Status.* A well-built man of 38, with no anatomical stig-

mata, and no external evidence of syphilis; he denied syphilitic infection, although he appeared quite frank about his sexual relations. Knee-jerks very much exaggerated, the left apparently somewhat more so than the right, but both were so active that it was difficult to come to a definite conclusion; Achilles-jerks exaggerated, the left more so than the right; the triceps reflexes much exaggerated, more so on the left side. The skin reflexes were active; plantar flexion on both sides. No motor weakness; gait normal; no sign of Romberg; slight ataxia in touching the nose with finger-tip, but no difference on the two sides; tremor of fingers and tongue. No disorder of sensibility. Spontaneous speech slow and deliberate; marked tremor, sticking and distortion in pronouncing the test phrases. Writing tremulous with occasional distortion of words, although he knew how to spell them, *e. g.*, "ateraley birgade" (artillery brigade).

Slight external strabismus of left eye with a faint variable indication of ptosis; pupils irregular, unequal, L. 5½ mm., R. 3½ mm., both show practically no reaction to light, both react defectively on accommodation, especially the right. No hemianopia. Marked lymphocytosis of the cerebro-spinal fluid. No disorder of sensibility. Internal organs satisfactory.

*August 4, 1907.* Since admission the patient has shown little change. He accepts the environment philosophically, although he has said once to his wife that he was disgusted to be with crazy men; he does not at all realize why he was sent here. Although he is rather anxious to leave, he finds it convenient to stay for a few weeks until his wife receives the proceeds of a sick benefit.

His physical condition is unchanged; there have been no neurological episodes and no headache except after lumbar puncture.

*On August 30*, the patient left the hospital. He was able to work for a few months, but later found no occupation. He had several attacks of twitching of the left hand and leg without loss of consciousness. He became somewhat forgetful and childish; he frequently came home cut and bruised, having apparently fallen. He was taken to Bellevue Hospital in April, 1909; there he was excitable and uneasy, now elated, now depressed without cause.

On re-admission to Manhattan State Hospital, April 6, 1909, the patient showed very little deterioration from his condition when first admitted to the hospital; his orientation was good, he had no absurd ideas, but showed the same cheerfulness as on first admission; his memory showed several discrepancies, *e. g.*, married at 20, 18 years ago, was now 40. He showed the same want of insight into the seriousness of his condition, denied that he was sick, thought that he could do his work better than ever.

*Physical Status.* Knee-jerks exaggerated, the left more so than the right; a tendency to clonus of the foot on the left side; plantar flexion on the right side, doubtful reaction on the left; no weakness

of the limbs of either side; slight weakness of the left side of the face; no sensory disorder, but the patient did not localize touches very accurately; tremor of tongue, fingers and facial muscles; sticking speech without distortion of test phrases; the writing was much worse than on first admission, he wrote " Medodist Epistal" "Tirth Riding Bigade." Pupils unequal (left greater than right), did not react to light at all, reacted sluggishly on accommodation.

During the summer the symptoms showed no progression.

*On August 18, 1909*, at dinner, the patient did not eat; he grasped the *left hand* with the right; he said that he felt all right and he was able to walk back to the ward. When seen after dinner he was still holding the left wrist tightly with the right hand. At 2 P. M. the left arm was continually twitching; he tried to control this with the right hand. He talked in an unintelligible manner, continually repeated "the bones wont blong kelasp." He moved his legs restlessly. His lips and tongue were in continual motion. He seemed to react less to pin-pricks over the left arm than over the right. During the whole attack he perspired freely. When examined at 7.30 P. M. no weakness of the left hand was demonstrated, but there was some impairment of sensibility. He did not feel light touches on the left hand, shoulder or face, whereas he felt them on the right hand and face. He did not recognize a watch or knife in the left hand; he immediately recognized them in the right.

On August 19, the patient could walk unassisted, but the left leg seemed slightly stiffer than the right; the left hand grip was a little weaker than the right. On August 28, lobar pneumonia of both lower lobes developed, and did not completely clear up; in September, the symptoms pointed to abscess of the lung. His mental condition showed no change; he remained quite clearly oriented, felt happy and contented, was optimistic as to recovery, said that he did not need to work, that he had enough money to keep him; his memory was no worse than on admission.

Until the day of his death the patient remained absolutely clear, and his memory showed no progressive impairment.

He died on November 5, 1909.

CASE 12. F. S., 54, admitted to Manhattan State Hospital August 4, 1909.

*Family History.* Negative.

*Personal History.* The patient was born in Dublin in 1855, developed normally, received a good education, became an expert tea-taster; he was a quiet, hard-working young man with an even temper. About the age of 20, he was thrown off his horse while hunting, was unconscious for several days. According to his account he had paralysis of the *right* side for three months; he lost his sense of smell and of taste and became deaf in the right ear. He had to give up his occupation and later came to Canada, married in 1877, then came to New York and worked as a bookkeeper. His disposition must have

changed after the accident, for, ever since his wife knew him, he was rather peculiar and violent-tempered. Not long after marriage his wife reproached him for coming home late; he took out a revolver and fired into the wood-work of the room. He would occasionally disappear at night; he would sometimes not speak to his wife for days; at other times he would treat her outrageously. He did not drink to excess and was quite tolerant of alcohol.

In 1896 (approximately) the patient had two epileptiform convulsions, after which he was in bed for a week. He behaved in a somewhat boyish manner with his male attendant. After this he was somewhat dull at business, fell asleep easily. He had epileptiform convulsions at long intervals, five in all, the last in 1908. For seven years previous to admission he had minor attacks; during an attack he would laugh foolishly, turned purplish white, tended to fall, his mouth worked, he would try to talk and when able to articulate would utter nonsense. In half an hour the patient would be as well as before the attack. He had sometimes three or four such attacks in one day. During the two years previous to admission, he was supported by his wife and accepted the situation placidly. His memory became impaired, he began to be careless in his dress; finally his gait became slightly altered and his speech was affected. At night he would behave peculiarly; he would light matches under the sheet in bed and would search under the bed with matches. Up to two months previous to admission, he was able to keep his wife's household books, but at the end he was no longer able to do this.

On admission (August 4, 1909), the patient behaved in a very gentlemanly manner and showed no peculiarity of behavior. He told spontaneously about his sickness and said that he was "losing active control of my brain, unconsciously, I should say, because I never know now-a-days whether I have had it or whether it is coming. My trouble is my want of memory and at times I am incapable of doing mental work, and lose all consciouness of existence." He gave a very fluent story of his life but some of his statements were false and in some of his dates he showed glaring discrepancies. He denied that he had any children; as a matter of fact he had a daughter alive. He denied syphilis, admitted gonorrhea at the age of 17. His grasp of general information showed some striking defects; he said that the battle of Waterloo was in 1854. His retention was rather poor.

*Physical Status.* No physical stigmata; no evidence of syphilis, nutrition good; knee-jerks and Achilles-jerks active, the right knee-jerk a trifle more active than the left (not confirmed on later examination); no motor weakness; no impairment of sensibility; complete anosmia; he was able to differentiate salt and sweet solution, but on later examination his sense of taste was found to be very defective; hearing in the right ear was impaired; pupils unequal, slightly irregular, reacted briskly to light but with a limited excursion, well on accommodation; very slight tremor of tongue and fingers; speech

slurring and sticking without much distortion of the test words; marked distortion of written words without tremor, *e. g.*, mesodeth epispocal (Methodist Episcopal); gait somewhat deliberate but otherwise not peculiar; no sign of Romberg; abundant lymphocytosis of the cerebro-spinal fluid; no disorder of the visceral organs.

In further interviews the patient varied considerably the story of his life. He soon found detention irksome and harped on going back to business.

*On September 14*, the patient suddenly became unsteady in his gait. When questioned by the attendant, he talked incoherently, he was dull and confused, he muttered a series of meaningless syllables. After an hour and a half he was in his usual condition. No other attacks were observed during the rest of the year.

In October he was allowed to go home, but, as he was quite unreasonable and difficult to manage at home, he was brought back in March, 1910. His wife reported that he would wander round the house at night, he would strike matches, look under the bed, shake the pillows; he would fail to recognize old friends; at times he could not find his own room; his memory of recent events was very poor.

On readmission the patient was noticeably more dilapidated than when discharged. He took his detention much more easily; his memory showed still more glaring discrepancies; his retention was very poor. At night he would be a little more confused and disoriented, but during special observation no minor attacks, similar to those described by his wife as occurring previous to admission, were observed. He never made any grandiose claims. The pupils now reacted very slightly to light, but well on accommodation; otherwise the neurological status was practically unchanged.

During the summer the patient became more slovenly and dilapidated.

*On November 10*, he fell on the floor unconscious; no twitchings were observed; sign of Babinski on both sides. The cerebro-spinal fluid was examined by the Noguchi modification of the Wassermann method and gave a positive reaction. The patient did not recover consciousness, and died on November 19.

CASE 13. P. D., 47, laborer, right-handed; admitted June 29, 1906.

*Family History.* The parents lived to an old age. No further information could be obtained.

*Personal History.* The patient was born in Ireland in 1859. He received very little education, but learned to read and write. Little information as to his early life was forthcoming; he himself said that he had been in the English Army for six and a half years and he talked of having served abroad. He came to America in 1883 and married in 1898. He was a temperate man; he denied syphilitic infection, and there was no external evidence of it. His wife had one child in 1899, then a series of miscarriages due to prolapse of the uterus.

The patient was absolutely healthy until February, 1905, when he

was struck by a trolley car, and was dragged a considerable distance; he received a severe scalp wound, and remained unconscious for four days. After two weeks he was able to get up. No weakness of either side was observed; he was able to speak quite well; he showed no delirious symptoms; for some time after the accident he was irritable and had severe headache; he lost all sexual desire, In June, four months after the accident, he returned to work and seemed absolutely efficient for some time.

Early in 1906, he began to show mental symptoms; he became forgetful, had especial difficulty in recalling names, was slightly peculiar in his talk, seemed to know what he wanted but could not make his tongue go right; he was impatient if the others did not at once understand. He was inefficient at work, and rather sulky in disposition. At night he would be heard talking as if expostulating with his fellow workmen; he heard their voices talking to him, and would even go to the door to see them.

*On March 21, 1906*, after confused behavior lasting fifteen minutes, he gave a cry, fell on the floor and had a series of three general epileptiform convulsions, beginning in the muscles of the face. After the second fit the whole right side was helpless. He was taken to a hospital where he was unconscious for four days. When he became conscious he spoke thickly and used wrong words, understood what his wife said, but was unable to express himself and was irritated by the fact that his wife did not understand him. In hospital, in addition to the right-sided hemiplegia it was noted that the right pupil was dilated, and that the right eye was not closed during sleep. He returned home after seven weeks, and still showed some paraphasia, but no apparent weakness of the right side. In the second week of May he went to work, but after ten minutes on turning round on a plank he lost his balance, fell and fractured the base of his skull. That evening in hospital he was wildly excited and remained delirious for several weeks; when the delirium subsided he was confused, stupid and demented; he answered questions apparently at random, sometimes became violent without apparent cause; he showed well marked euphoria.

*On admission* to Manhattan State Hospital (June 29, 1906), the patient talked to his fellow patients in a somewhat confused manner, at times he laughed, at times he cried without apparent reason, and frequently he got out of bed and wandered around in an aimless manner. When interviewed he was good natured, and much interested in the typewriter. He occasionally answered a simple question relevantly, but as a rule he did not seem to understand the questions, and answered them in a quite irrelevant and sometimes meaningless manner with marked perseveration.

Have anything to eat to-day? "Oh, yes; breakfast."

What did you have for breakfast? "I generally have bread and butter and tea."

What is your name? "Spit (a patient is spitting)—spit out."
Who are you? "Spit—no, I don't spit."
Tell me what you are? "P. D." (his name).
What is the name of this place? "Spit clean out is the way they do."

He showed similar perseveration on the words "rough" and "Irish." Is it summer or winter? "Yes, that is the way—they would call that rough."

Owing to this aphasic difficulty of understanding and of expression it was impossible to ascertain definitely his orientation, the state of his memory, his grasp of general information, and whether he had any morbid ideas or not.

*Physical Condition.* The patient was a well nourished, robust man, with no anatomical stigmata, and no evidence of syphilis. There was a linear scar over the right eye and over the right occipital region with a slight depression of the skull. The pupils were unequal, irregular, did not react to light, reacted extremely defectively on accommodation. He walked in an ataxic manner. Absence of knee-jerks and Achilles-jerks; speech slow and hesitating; writing very tremulous and with marked distortion of words. He wrote his name as Patr14y (Patrick). There was coarse tremor of the fingers. The hand grips were equally powerful; movements of the face were symmetrical; there was no weakness of either leg. No sign of Babinski. Sensibility to pain was apparently normal. He did not co-operate satisfactorily when his sense of touch was tested.

One week after admission the aphasic symptoms were less marked; the patient answered more relevantly, and did not show the same perseveration.

*On July 10*, at night, he had several convulsions which were confined entirely to the right side, the face being especially affected. On July 11, he showed complete inability to talk; his lips moved, and he made futile efforts to talk; it was not noted whether he understood simple commands. There was weakness and impaired sensibility over the right face and right arm, the latter being rather spastic; the right leg was not affected. The duration of these symptoms was not noted.

*On July 20*, he had a convulsion which seemed to leave no residual weakness; the condition of his speech was not specially noted. On August 1, at night, his right arm was noticed to be rigid, but no convulsion was observed; in the morning the right hand was weak and spastic. Examination of his speech on this date disclosed great difficulty of expression; sometimes his answers were unintelligible, usually they were irrelevant, and frequently consisted of "I couldn't tell you." He was unable to name objects, muttered unintelligible syllables; he recognized the correct name of a pencil when he heard it.

*On September 17*, he had a series of convulsions, during which the movements were limited to the right side; between the convulsions

he was conscious. For some time after the last convulsion he was unable to speak. His gait became worse so that in October he was put to bed. On October 3, his condition was noted as follows: The patient apparently is unable to comprehend questions, and his answers are entirely irrelevant; when shown different objects, he calls them all a "whiska" and when asked questions his answer is always the same.

An examination of the aphasic condition was made on October 25. During the interview the patient laughed rather explosively from time to time; at other times, usually when he had tried to say something, he became very much irritated, shouted angrily, ground his teeth. He frequently did this when cared for by the nurses. He was quite unable to express himself, although he had occasionally been heard to make a definite remark, e. g., "What the Hell brought you?" He said "yes" and "no." In answer to questions he uttered unintelligible jargon, e. g., "bay – or – one – – – one – ef – one seef." He appeared to make great efforts to express himself, and sometimes was much irritated over his inability to do so. He could not be brought to repeat phrases or name objects or pictures.

He did not recognize spoken nor printed orders, nor words. In using objects he showed some asymbolia, tried to scratch a match upon a candle, fumbled with a pipe and was about to put it in his bed-urinal, put the thumb and index of the right hand in a peculiar manner into a glass of milk held in the left hand.

*Physical Condition.* (October 25): Slight general spasticity; knee-jerks and Achilles-jerks absent; pupils small, unequal and with no reaction to light nor on accommodation; right-sided ptosis. Slight weakness of the right side of face. There was no weakness of the right hand-grip (on the previous day some slight weakness seemed present), but he used the right hand spontaneously less than the left, and was slower in innervating it although the final grip showed no weakness; he had much more difficulty in picking up a pin with the right hand and occasionally let objects fall. He could hold a pen and wrote an unintelligible series of tremulous and distorted letters, whereas on admission he had been able to write an approximation to his name. He showed very little confidence in the upright position, although he was only slightly unsteady; sign of Romberg was present. He walked with his feet widely separated and his gait was spastic rather than ataxic. Tremor of hands and slight tremor of tongue. Slight general diminution of pain sense, except over face. Abundant lymphocytosis of the cerebro-spinal fluid. No disorder of the internal organs.

*On October 26*, he had six convulsions; the last two were right-sided, the others general; after the convulsions his general condition was unchanged.

The extent of the aphasia was not constant. On October 27, he made an occasional appropriate reaction to a spoken demand, but uttered nothing except "red – red" and a few simple words such as

"this," "I have"; on October 29, he gave no sign of understanding spoken words. On October 30, he made an occasional correct reaction, e. g., closed his eyes on demand, and said "that's right" when his name was mentioned. On November 4, he made no remarks to his wife but said, after an effort, to the physician "that is–my–wife," and two days later when asked how he was, he answered explosively "first class."

After November he was not heard to utter anything beyond "yes," and an occasional poorly pronounced "son of a b – – –."

The minimus and fourth finger of the right hand began to show some contracture in November. On December 4, the patient had four convulsions with twitching of the eyes and right side of face (legs not observed); after this attack there was transitory limpness of the right leg and arm.

In December, the contracture of the fingers of the right hand had increased, and the patient would open it with the left hand after considerable effort. There was no weakness of the right hand and it was withdrawn from pin-pricks. Plantar flexion on both sides. He put out his tongue when asked by gesture to do so; he carried out no spoken commands, showed no sign of understanding any words.

*On February 1, 1907*, he had continuous convulsions in the morning with jerking of the right extremities. On February 21, he had several general convulsions, after which the right face was twitching, and the right arm and leg did not react to pin-pricks while the left did.

During the Spring the patient became progressively feebler; a deep bed-sore developed; the right leg was much drawn up, the left was extended; both arms were held flexed on the chest, the right more rigidly than the left; the face was pulled to the left. The plantar reflex continued to be flexion on both sides.

He died on May 3, 1907.

CASE 14. M. H., 46, right-handed, widow; admitted to Manhattan State Hospital, September 5, 1903.

*Family History.* Both parents had asthma; otherwise the family history was negative.

*Personal History.* The patient was born in Ireland in 1857; came to the United States at four, received a meagre education, worked as a domestic servant. In 1878 she married an alcoholic painter, had four children, two of whom died in infancy. The patient had no miscarriages, there was no history of infection. The husband died in 1886. The patient worked in a laundry steadily till 1901 and was then supported by her daughter. She was a cheerful, healthy woman. During the year preceding her admission the patient had shown mental symptoms. She was fretful and irritable, would repeat herself frequently in conversation, tended to exaggerate. The menopause began early in 1903, and during that year the patient complained of severe headaches, which occurred once a month.

*On July 10, 1903*, the patient had a slight *right-sided* attack; at 6 A.M. she was found sitting on the bed moaning; she said she had a cramp in her right leg and hand, the hand and fingers were flexed and seemed to be stiff for two or three minutes. After being rubbed she seemed quite well, and after one hour she was able to do her work as usual, using her right hand freely.

On August 31 she complained of being dizzy. After lunch her right arm seemed to be again cramped and flexed as in the former attack. She said "Look at my poor hand." She wandered about as if confused and was put to bed for a couple of hours, her right hand and arm seemed to be lifeless; later in the afternoon she was able to use the arm; she appeared to be dazed; talked of being in a strange house; she vomited several times. Next morning she seemed to be quite clear, but during the forenoon she became delirious, screamed as if in fear, was very much excited; she was able to use both hands freely. She was taken to Bellevue Hospital. There she was disoriented, misidentified those about her, was rambling in conversation, talked of seeing imaginary objects.

On admission to Manhattan State Hospital (September 5, 1903) the patient was quite composed and reacted quite normally to her environment. Her mood was one of complacency; she frequently laughed in a rather simple manner; there was no evidence of hallucinations and the patient uttered no absurd ideas. She gave a clear and fairly coherent account of the onset of the sickness, telling of the paralysis, the arrival of the priest and the doctor; she knew that she had been taken to Bellevue Hospital. "There they said I seen something like rats or squirrels or something and that I hollered, I didn't know where I was when I went to 26th Street. The next day I was all right." She could not tell the name of the place but said that it was "for people who don't know nothing." She knew that she was in New York, gave the year as 1891, gave the day of the week correctly and said that it was the middle of September. She was able to give a fair account of her life—came to the United States at five, went to the Sisters' School until thirteen, then lived at Chicago with her parents, worked in an hotel, married at 22 in January, had her first child two days before Christmas. She had four children, no miscarriages, no still-births. Her husband had died seventeen years previously. She denied any venereal disease or secondary symptoms. She had a good memory of events up to the onset of the psychosis, remembered the arrival of the doctor and priest, but had completely forgotten the events of the following two days until she came to herself in Bellevue Hospital. "I then got up and went around and I thought it was something like a squirrel running around biting a piece off my skirts." The patient had a satisfactory grasp of the trip to the hospital. She was unable to retain a test number for two minutes; she could not retain the name of the physician or of the hospital although it was repeated again to her. Her grasp

of general information was extremely poor. She could only say the first part of the alphabet, could not name any large cities in the United States, was unable to do five times six, although she did one or two simple multiplications correctly. The patient realized that her mind had been affected in Bellevue Hospital and was willing to wait for her daughter to come and take her home. "I was sent here but I aint crazy, if I was, I would be hollering and carrying on."

*Physical Status.* The patient was a well-nourished and well-formed Irish woman with no external signs of syphilis. The knee-jerks were both exaggerated, the Achilles reflex well marked; the triceps reflex was well marked on both sides, the *right* seemed to be a little stronger; the plantar reflex was very faint on both sides, scarcely any movement of the toes being elicited. The tongue deviated slightly to the right, the right hand-grip was strong but perhaps a trifle less than the left; the facial movements were symmetrical; there was no weakness of the lower extremities; the gait was unimpaired; there was no sign of Romberg. The pupils were small, equal, reacted to light sluggishly and within narrow limits, reacted more promptly on accommodation. Marked fibrillary tremor of the tongue, occasional flickering of the facial muscles, fine tremor of the hands. As a diagnosis of hysteria had been made previous to admission, a very careful sensory examination was made so far as her somewhat unreliable answers made it possible. There was general diminution of sensibility to pain; over several small areas, two inframammary, two scapular, there was complete analgesia and anesthesia with dulling of the temperature sense. The speech was slurring with distortion of the test phrases. "Third ridin billery betrade." "Elec-crisity." She wrote her name with difficulty, distorted a test word beyond recognition.

During the first two weeks in the hospital, the patient showed practically no change. Her retention continued to be very poor; she showed some glaring discrepancies, when asked to give exactly the data of her life. She gave the year as 1892, said that she was 56, born in 1857. She could not correct this discrepancy,

*On September 27*, at 6 A.M. the patient was found sitting on the edge of the bed; the right arm was lying limp; she made an effort to speak but could not be understood. She beckoned to the nurse with the left hand. No twitchings were observed. The patient lay quietly in bed; she appeared to take no notice of anything going on. She showed a persistent tendency to look to the right. At 9.30 A. M. when examined, the patient gave no evidence of understanding commands and made no attempt to speak except when pricked by a pin. She then said "Oh, sir!" Her right arm was limp; the right side of the face was smoother than the left; the forehead wrinkled symmetrically on frowning. She was able to walk slowly without any dragging of either leg. There was marked diminution of the pain sense over the whole of the right arm, no diminution over the left arm; no difference in the reaction of the two legs could be made out. The deep reflexes

were more active on the right side than on the left. No sign of Babinski. The eyes were turned to the right, and showed some nystagmus. The pupils showed a faint reaction to a strong light; the patient did not wink in reaction to feinting from the right side, but reacted to feinting from the left. At noon the patient spoke a few words and made fumbling movements with her right hand. In the afternoon she recognized her daughter and named her correctly.

*On September 28*, there was no definite residual of the weakness of the right hand, but the patient at mealtime used the left hand, in preference, and fumbled with the right hand. The right side of the face showed fewer wrinkles than the left. The tongue deviated to the right. The right arm appeared to be somewhat less sensitive than the left. The head and eyes were no longer turned to the right. When brought into the room for examination, she said "Rought me in," but made no further effort to speak spontaneously. She showed no evidence of understanding spoken commands except when told to sit up. She did not pick out any objects named, did not appear to recognize such sounds as the ringing of a bell, the ticking of a watch; she merely said "I can't." She did not seem to recognize colors when named, and when shown pictures she talked in a *paraphasic* manner, e. g. (picture of kittens) "Oh! I wouldn't be sighing, I am sane, sane (? sayin')." She could not be brought to repeat simple words nor familiar series, such as the numerals. She could not name objects, simply uttered *meaningless jargon.* On the same day, the patient seemed to pick up something imaginary from the cover with the right hand, and dropped it over the bed. During the following few days the patient began to understand and use more words. She showed marked paraphasia and perseveration. At first she could not write spontaneously nor copy, but gradually her writing improved so that she could write an approximation of her name and address. The patient showed some jerking and fumbling with the right hand, especially when asked to write. On the evening of October 2, she appeared frightened, appeared to fling imaginary things from her right hand, said "Take it away, it is dirty." She continued to do this for half an hour. On October 3, she again appeared to fling something from her right hand in an apprehensive manner. On October 5, the patient appeared to be in practically the same condition as before the attack. She was able to speak freely and gave an account of the onset of the attack. "I took sick in the morning I think, it seemed to me I couldn't speak and I didn't know them, when Ada and my son-in-law came, I didn't know them." She had apparently a very hazy memory of her behavior during the past week, and no memory of having behaved peculiarly or in response to hallucinations. Examination of the cerebro-spinal fluid on October 25 disclosed a pronounced lymphocytosis.

During the following six months the patient showed little change and was allowed to go home on April 19, 1904. Her conversation dur-

ing the three days at home was continually about the hospital. On the fourth day at breakfast time, she was unable to use her right hand and said "Bad luck seems to fall (follow?) me." She was placed in bed; two hours later she was shouting excitedly the names of people and appeared to be in a delirious state. She was taken to Bellevue Hospital; there she was rambling and incoherent and laughed childishly.

*On April 27* she was readmitted to Manhattan State Hospital. Her physical symptoms were the same as when discharged from the hospital. The right side of the face was slightly flatter than the left. No gross weakness of either arm or leg was observed; the right elbow-jerk was more active than the left; the knee-jerks were exaggerated but no difference was observed between the two sides; no sign of Babinski.

*On May 9*, the patient went to the clothes-room, forgot what she wanted, sat down on a chair; she was pale, but was able to walk with assistance to her bed. When examined five minutes later by the physician she apparently made an effort to talk but said nothing. She opened her mouth on request but did not protrude the tongue. There was no twitching. The muscular condition on the two sides appeared to be the same. Pin-pricks caused no movements on the right side, but were reacted to over the left side. The pulse was 64. The color slowly came back to the face. Half an hour later the patient had a similar attack. When observed a few minutes later, she was making purposeless movements with the right arm, head and eyes. These movements soon ceased. Pin-pricks over the *right* arm, chest and abdomen caused no movement; pin-pricks over corresponding areas on the left side were definitely reacted to. Pin-pricks over the right thigh and leg were reacted to but not so promptly as over the left leg. Plantar reflex; flexion on the right side, not determined on the left side. The patient had nystagmus for an hour and a half. The pulse was 60; four hours later it was 100.

*On May 10* the patient showed marked paraphasia. She appeared perplexed and somewhat apprehensive The duration of the paraphasia was not noticed.

Towards the end of June, the nurse observed that the patient frequently had attacks of dulness, when she would, for a few moments, be unable to respond and appeared not to understand what was said to her. At these times her right arm would frequently appear to be more or less helpless. These attacks would often be noticed when the patient awoke from sleep, or they would be preceded by moaning for a few minutes. The patient's mental condition showed considerable deterioration, she knew where she was, but had no idea of the month or year. She grasped questions with difficulty. Her answers were rambling and full of repetitions. In what year were you born? "I can't tell, tell what year I was, I can't tell where, she's 23, I was 20, no 23, she said she is 23 – –." When asked to write she was

unable to grasp the pencil suitably without help; she made a few awkward lines. She then seemed to become stupid, the right angle of the mouth and the right thumb twitched; the right arm was found to be limp and helpless. She did not appear to understand what was said to her, answered "no" and "I can't" to all questions. After about ten minutes she was much brighter, was able to move the right arm. She did not comply with simple demands, she called a watch a "clack"; (pencil) "that's I know what that is, a writing, t'is". The patient did not appear to notice objects in the right field so readily as in the left. Sensibility was duller over the right arm than over the left. There was no gross motor weakness on the right side but movements of the right hand were more awkward than those of the left.

*On June 27*, there was transitory weakness, or, more accurately speaking, want of utilization, of the right arm; she used the left hand by preference, the right arm seemed helpless, but she was able to use it freely when urged to do so.

*On June 28*, she had two minor attacks. In the first, which lasted only a few minutes, the head and eyes turned to the right, both arms twitched, the right more than the left; then she began to fumble with left hand, and was able to answer "yes" and "I don't know". Two hours later she had another attack in which the right face and arm twitched; no weakness of either hand-grip was observed after this attack.

The patient had no attacks in July; she understood well what was said to her, was able to name objects correctly; the right hand was a little weaker and less steady than the left; no sensory disorder was demonstrable.

*On August 5*, she had a transitory weakness of the right arm, was weak, tended to fall to the right, appeared dull and vacant; in a few minutes she had returned to her previous condition.

*On October 9*, she had an attack of dulness, with accentuation of her speech defect; she complained of her right hand, but no gross weakness could be demonstrated (co-operation poor).

The patient showed progressive deterioration; in November she uttered absurd ideas about her dead husband having returned alive. The pupillary reaction had become more defective; the left pupil did not react at all to a strong light, the right showed only slight reaction.

*On December 16*, after several days' drowsiness and reduced talkativeness, she was, in the morning, unable to utter articulate sounds; in the forenoon she could say "yes", "no"; she could not use her right hand. In the afternoon she was able to speak, but rambled on in a fragmentary way about her work in the laundry, showed some difficulty in finding the right word. She recognized objects, had difficulty in naming them, showed paraphasia and perseveration. (Watch) "Courses, good courses, corset, silver corset." (Two dollars) "That's nice, two chisl, one corset, it's a corset, you have that

to spend two corset." She did not carry out simple spoken orders, but answered some simple questions after they were put several times. She was able to imitate various gestures shown her. She wrote the initial letter of her name, then stopped. No difference between the knee-jerks could be demonstrated; ankle-clonus was present on the right side, but only occasionally was there a tendency to clonus on the left side. On the following day the patient was able to use her right hand.

In January, 1905, she had deteriorated further; she mistook her daughters for her sisters, immediately forgot that she had had a visit. She showed fair understanding of simple questions and commands but marked paraphasia and perseveration with senseless reiterations. The right arm showed no gross weakness; the right elbow-jerk remained stronger than the left. In walking the ball of the right foot scraped the floor a little. Plantar reflex, as a rule flexion on both sides with occasional doubtful extension on the right side.

During 1905 the patient showed progressive deterioration; she soiled the bed continually. No weakness of either hand-grip was noticed, but the right hand was usually held in the left hand or on the chest. In walking the right foot rubbed the floor slightly. The difference in the reflexes of the two sides was slight, the right being the more active; ankle clonus and plantar flexion on both sides; the ankle clonus was more easily elicited on the right side. The pupillary reaction to light was lost. She was quite unable to write even an approximation to her name; she made meaningless scrawls. She reacted to pin-pricks over all parts of the body.

In February, 1906, the patient was now too weak to walk to the chair. No further neurological incidents occurred. In March broncho-pneumonia developed and the patient died on April 3, 1906.

CASE 15. W. S., 49, laborer, right-handed; admitted July 8, 1905.

*Family History.* A paternal grand-aunt was insane in advanced life; the mother was silly and simple for a few weeks before death at 68. One brother of the patient is alcoholic and somewhat abnormal.

*Personal History.* The patient was born in New York City in August, 1855. He was probably neurotic as a child, received a fair education, took up work in a dry-goods store. He was cranky and irritable, indulged to excess in alcohol and tobacco; his sexual habits were not known. He married at the age of 34, and his wife had one boy of a rather nervous temperament, no miscarriages; he did not wish to have any more children. In 1902 he lost a position as assistant-foreman in the street-cleaning department, took this very much to heart; he had to take a job as an ordinary sweeper. In 1903, he became more irritable than usual, ill-treated his child; in 1904, *his speech became defective*, he would start to tell something and stop in the middle; he showed peculiar behavior. One day towards the end of 1904, he was unable to speak for ten minutes, although conscious.

In December, 1904, when brought home after a convulsion on the

street, he could barely articulate and mixed his words up, bringing them out with great effort; he would scream out his words in the effort to speak; he was able to understand the doctor's requests; he was temporarily unable to read or write; after two days he could read and could soon write again (wife's account). He resumed work in January, 1905, but frequently told of spells during which he had become speechless and had to sit down. His wife frequently observed such attacks in the house; he could not answer any questions, would utter an inarticulate groan; as soon as the spell was over he was able to go out, but for a short time the right arm would be weak; he would be unable to lift his cup. At times he complained of numbness in the arm. These spells occurred several times a week; his gait, even between the attacks, was staggering, although he did not seem to notice this. On one occasion the patient lost his way in town and could only give a very hazy account of his wanderings.

About three weeks before commitment he had a very severe attack, and after this he began to act outrageously towards his wife and child; for two or three nights before his removal to Bellevue Hospital he talked of people coming to throw him into the boiling fat, he said "take these dead bodies away."

In Bellevue Hospital he was quite disoriented, thought that he was in a dry-goods store, and that he had been there for three weeks (two days); he gave the date as January, 1904, said that he felt fine. He talked continually at the top of his voice, was restless and irritable.

*On admission, July 8, 1905*, the patient was confused and restless, picked at the bed clothes, made feeble efforts to rise; he grasped aimlessly at the physician's hands, answered questions in a tremulous, stammering voice. He resisted the care of the nurses in a rather turbulent manner, and was placed in a tepid pack for several hours.

When examined on July 10, he showed extreme mental dilapidation; he seemed to have no idea of the environment, he did not recognize the physician as such, he could not tell when he had come to the hospital. His mood was one of complacent good humor.

*Physical Status.* General nutrition fair; no external evidence of syphilis, but the glans penis could not be examined owing to phimosis; Argyll Robertson pupils; knee-jerks much increased; Achilles-jerks not elicited; plantar flexion on both sides. No weakness of either side. Gait ataxic; sign of Romberg. Tremor of face and tongue; speech and writing very defective.

In the months following admission he had occasional attacks of weakness, and in September he had a convulsion. In October he became so ataxic that he was placed in bed; at this time his pupils were definitely unequal. When examined in November he was unable to write his name, could only scrawl a few letters.

*On December 17* the patient had an attack of weakness, collapsed toward the right side, but grasped well with the right hand; it was difficult to loosen his right hand from the bedstead which he had grasped;

the right leg was somewhat flexed, and if touched was withdrawn and began to twitch; he kept the right leg somewhat flexed; the right foot would twitch as soon as it touched the floor. He could not be brought to say more than "yes." *On December 18* he named objects incorrectly and showed perseveration: (Pencil) "Pencil." (Eye-glass case) "Comb". (Eye-glass in case) "It's a spoon" (Eye-glass) "It's a spoon" (What for?) "We comb with that".

This difficulty in naming objects persisted for a few days and he showed marked paraphasia, *e. g.*, December 23, (Pen) "That's a le-le-ste lencil." (Pencil) "That's because for co-coa many a shawl—." (Keys) "Oh go-go-go-go—I cal them ca-casmus ay—I call them castles". On a later attempt he named them correctly. In speaking he became a little excited and poured out a series of meaningless syllables with energy. His spontaneous utterances consisted of a disconnected series of words and phrases. On this date there was no weakness of the right hand-grip.

The difficulty in naming objects was not observed in January, 1906, but in speaking he frequently stuck and stuttered, so that many utterances were quite unintelligible. *In February* he spoke more freely than previously, was much brighter and walked better; he said: "Thank God I'm getting stronger and won't die here". There was quite a contrast between his comparatively free delivery and his previous drawling, sticking and stammering. He could now write his first name, but was unable to complete his second name. He said—"Isn't it fierce I'm nervous, I am a d—d fool to write like that". He was able to give a much better account of his life than at any previous time in the hospital; he said, "to my knowledge it is a place for you taking stating (statements—physician's notes) for outside jobs—for taking examinations for public jobs." He had no idea of the season or year. He denied that there was anything wrong with his mind. In March he had returned to his previous low level; in May he had a sudden transitory attack of unconsciousness with an unexplained temperature of 103°; the eyes moved restlessly; no difference on the two sides was observed, except slightly greater rigidity of the left arm. Two days later the temperature again rose for two days and then returned to the normal.

During the summer the patient showed progressive enfeeblement without any further neurological incidents; an axillary abscess developed and he died on September 18, 1906.

Case 16. M. L., 33, right-handed, plasterer's helper; admitted May 1, 1905.

*Family History.* One maternal cousin was insane and died in an asylum. A maternal uncle died suddenly at the age of 14, from some brain trouble. The patient's wife thought that other relatives had been insane, but no definite information could be obtained.

*Personal History.* The patient was born in Ireland in 1872, was a bright child, learned to read and write, worked later as a laborer,

came to America in 1890, married in 1899. Nothing definite was known as to venereal infection except that the patient had admitted to his family doctor that he had had syphilis; he was always alcoholic, and during his married life drank beer and whiskey to excess. His wife knew of one mild delirious episode, but denied that he had ever had delirium tremens. For ten years previous to admission the patient suffered from headache and occasional neuralgia. In 1903 he began to drink less in order to save money. The exact date of the onset of symptoms could not be determined, but in 1904 he behaved peculiarly at his work, would stand looking vacantly or fixing his buttons instead of working; his comrades thought he was under the influence of drink.

*In September, 1904*, he one day came back from work and said, "I do not know what happened to me to-day, I can hardly get the words out;" it took him a considerable time to articulate, and his wife understood him with difficulty; no motor weakness was observed. He was able to work next day and his speech improved for a time, but in December became worse. In November his gait had become very unsteady; he would stagger when going across planks, was unable to keep a job more than a week. He was very irritable, treated his wife and children harshly; he was untidy, would spit on the floor, occasionally passed water in bed. During the winter he was sleepless and restless, occasionally arose at absurd hours. He showed no shame in exposing himself before the children.

*On March 21, 1905*, he lost his way outside, but was cunning enough not to let some acquaintances, whom he met, notice it; on the same day he could not sign his name on a check, he said that his arm was all right, but that he had forgotten how to spell his name. As he recognized that his speech was impaired, he was coaxed to go to Bellevue Hospital to consult a physician. He there said that there was nothing the matter with him, except that his voice was not good, and his memory somewhat defective; he gave the date as 1885.

*On admission* to Manhattan State Hospital (May 1, 1905), the patient was quiet and agreeable, readily entered into conversation, had no morbid ideas of greatness nor of persecution. He gave the date correctly, although he occasionally gave 1895 for 1905; he did not know exactly where he was, described the environment vaguely as "a nice place." He could not at first give the name of the hospital from which he had come, but later gave it correctly. He gave correctly his age at marriage, and the duration of his married life. He could not retain a name or number given him for ten minutes. He showed extreme ignorance of general information, although he had been to school for several years; he repeated the alphabet incorrectly, took thirty seconds to complete it. He had no insight into his sickness; "I am strong and healthy, the only thing the matter with me is my voice."

*Physical Condition.* A well nourished man who admits syphilitic

infection. Pupils unequal and with sluggish reaction. "Speech very ataxic." Knee-jerks exaggerated.

During the summer the patient showed only slight mental decline. He could name the hospital correctly, but did not realize that his fellow patients were mentally affected; he would give the year as "1995 and one", said that it was February (October). He could give a fair outline of his life but became hopelessly confused in an attempt to give accurate dates; he had come to America at 28, at 24, at 22, at 20. He was 22, 26, when he married. His mood was one of complacency, with occasional tearfulness over detention.

*On November 1*, about 11.30 A. M., he was observed to be walking unsteadily, dragging his *right* leg. Examined at 11.45; on the right side—sign of Babinski, ankle clonus; on the left side plantar flexion, no clonus; knee-jerks equally exaggerated. At first he gave an equally good grip with both hands, but was much more *awkward* in grasping with the right hand. When placed at a table in order to write he said that the *right* hand felt funny, he raised it with the left hand, dropped it on the table to limber it up. When asked to give a hand-grip he was now unable to grip at all with the right hand. The tongue was put out straight. He showed a little difficulty in naming objects, with slight paraphasia. Paper: "It's - - - - I can not say nothing." Bottle: correct. Paper: "Bottle—water (laughs at his mistake) oh! water." He said of his hand, "now it's *niff* (stiff)." He seemed confused; when asked where he was he grinned, but gave no answer. A few minutes after the examination he was taken to dinner; he took his spoon in his right hand, but used it awkwardly, used his left hand for his meat. He did not name the various dishes when asked. At 1.45 P. M. he cheerful greeted physician, said: "I'm all right now, doctor." No trace of right-sided weakness nor clumsiness, nor of speech defect (except his usual articulatory disorder) was now observed; he was able to write. Plantar flexion on the right side; no clonus. He remembered the incident well, said that for a quarter of an hour he had felt quite shaky and did not know where he was. "My feet were queer—my feet got stiff—my legs too."

His general physical condition at this date was as follows: General nutrition excellent; pupils irregular, unequal, reacting promptly but not extensively to light; patellar and Achilles-reflexes exaggerated and equal: upper tendon reflexes very active; writing tremulous, irregular with marked omission of syllables and distortion of words, *e. g.*, methese espeseaes (Methodist Episcopal), 1 nveere (November 1); speech slurring, sticking and rather staccato but without omission of syllables; internal organs satisfactory.

*On December 17*, the patient had a similar transitory right-sided attack. He said in the morning that he felt queer and staggery; his right hand seemed powerless, he held it in his left hand; he could just fold his right fingers round the nurse's hand without gripping;

he said that his right leg was weak. He stammered a good deal when talking, and the muscles of his face twitched. When examined by the physician at 9.15 A. M. there was no weakness of the right arm or leg, no sign of Babinski, no difficulty in naming objects.

During 1906 the patient had a variety of transitory attacks, and his mental condition gradually became worse. No special decline was seen in relation to the neurological episodes. He treated all these incidents as of no importance, and was annoyed when they were emphasized.

*On January 9, 1906*, he was seen to be stamping his legs; he said that both felt numb and that there was a numbness of his *left* wrist; no weakness of hands nor of legs; plantar flexion.

*January 14*, sudden weakness of the *left* hand leaving no trace in three minutes.

*January 25*, during the night he had transitory stiffness of the *left* arm; he left his bed, staggered about the ward, tumbled over a chair; he began to cry, said, "I don't know what's the matter with me."

*March 20*, he had a transitory weakness of the *left* side lasting for ten minutes; the leg was weak, the leg and arm felt stiff, the left cheek was flattened and moved less than the right. No objective sensory disorder was made out.

*March 21*, transitory numbness and weakness of the *right* hand.

*November 16*, he fell on the floor of the bath-room, received a scalp wound over the occiput.

*November 17*, the patient went out for exercise and had been talking as usual; at 10.30 A. M. he was found sitting on a chair with the *right* hand in the left; the right side of the face was smooth, the mouth pulled to the left; the right leg was dragged on walking. When asked how he was, he said "all right", but answered no other simple questions about his condition; he carried out no simple commands, merely looked stupidly in front; the lips and tongue were constantly moving. After ten minutes he answered his name. How old? "Over – for – or – or"; no intelligible sound. Name of this place? Unintelligible mumble. He could not name objects shown. Fifty cent piece: "I forget now." Keys? No response. Cent? He took it in his hand, rubbed it over his forehead, made the sign of the cross, began to mutter a prayer (? Asymbolia). Plantar flexion on both sides; no ankle clonus; knee-jerks both much exaggerated. At 11.45 the right leg was still weak, the hand-grip was better; he showed much less difficutly in naming objects. Next day there was no residual from this attack.

During October and November the patient became much more demented, less anxious to go home, more pleased with the hospital; his speech was almost inarticulate.

*December 3*, the patient became extremely weak, barely able to walk, was placed in bed. Next day he was in his usual condition.

He became progressively more stupid, failed to recognize his nieces and his brother-in-law; he called the charge nurse his wife's husband; he was able to give correctly the name of the hospital even at a very late stage of the disease. He said it was summer although the ground was covered with snow (January 28).

During the Spring he became feebler; broncho-pneumonia developed. He died on April 15, 1907.

CASE 17. A. H., 52, musician; admitted to Manhattan State Hospital, March 21, 1907.

*Family History.* Negative; information very defective.

*Personal History.* The patient was born in Hungary in 1855, developed normally and received a good education. His father was a musician; he himself early showed musical talent, and became a musician of the cafés. He drank to excess, indulged in venery, received some venereal infection as to which he could give no trustworthy information. He married at the age of 22; his wife had two children, no miscarriages.

He came to America in 1890 and played in various hotels. After several years he became rather short of breath. More than a year before admission he felt sudden precordial pain one evening; from this date he was unable to continue playing. He consulted a physician and received treatment at home; he was sleepless, restless, without appetite; he complained of a burning feeling in his head; he could not support any noise. He became progressively more apathetic, but showed no evidence of morbid ideas nor of hallucinations. His wife could not support him, and he was committed on the ground of his mild dementia.

*On admission to Manhattan State Hospital* (March 21, 1907), the patient was dull and apathetic, he accepted without comment the change of environment, was quite accessible when examined. He knew where he was, but could not give the date. He showed marked memory defect, made contradictory statements about the ages and number of his children, his own age, and various periods in his life. He knew that he was sick. He gave no evidence of any morbid beliefs, had no expansive ideas, was pleased with the environment and rather more cheerful than his situation warranted.

*Physical Condition.* A well-built man with no anatomical stigmata; history of syphilis at 28 (patient's statement). He complained of headache and dizziness. Pupils irregular, reacted on accommodation; the right did not react to light, the left reacted very faintly. Knee-jerks and Achilles-jerks absent; gait and upright position unsteady; sign of Romberg. Slight general diminution of pain-sense; touch and localization satisfactory; no local differences. Tremor of tongue; speech slow and deliberate and with distortion of long test phrases. Heart somewhat enlarged; no valvular lesion; definite arteriosclerosis. The sudden onset of cardiac pain one year previously suggested the presence of coronary sclerosis.

*On April 1*, after being on the chair for some time, he could not rise, the *left* hand was quite limp, he could not advance the left leg, required to be carried to bed; he did not answer questions, but appeared alert, took liquid nourishment well. A few hours later he answered questions relevantly, but showed definite *weakness of the left face, arm and leg;* plantar flexion on both sides.

*April 2*, the weakness still persisted, but he used his left hand in gestures and was able to give a hand-grip; there was no special sensory disorder on the left side; he lay looking to the right side, he did not wink when feinted at from the left side; he paid no attention to movements nor to an electric light in the left field. *On April 3* there was no trace of hemianopia.

*On April 4*, he showed a peculiar transitory motor disorder of the *right arm;* at noon he was unable to feed himself, could not pick the spoon off the table, made wide ataxic movements missing the table altogether; his right hand would go under the table or widely over it; he grasped his bowl by the far side, spilt the soup. When examined three hours later there was no trace of this ataxia of the right hand; he was able to take a cup of milk in the right hand and drink it without spilling it.

When placed on his feet his left leg gave way entirely and he had to be put back to bed. He now complained of weakness in both hands, but would not co-operate when his strength was to be tested. He pointed to his *left* hand as if it felt peculiar. The test for hemianopia was inconclusive, but he did not react so definitely to feinting from the *left* side as from the right.

*On April 8*, he was dull in the morning, alert in the afternoon; it was more difficult to persuade him to grip with the *left* than with the right hand; both hand-grips were weak. Plantar flexion on both sides. No difference in the reaction to pin-pricks on the two sides. He did not react when the eyes were feinted at from the left, reacted when feinted at from the right; he did not follow objects in the *left* visual field, followed objects in the right field. There was evidence of pulmonary congestion; P. 136, R. 40, T. 103.2°.

*On April 10*, he was bright, but had no memory of his wife's visit on the previous day. He said that his *left* hand was weak; it appeared somewhat weaker than the right, but he gripped sufficiently firmly with the left hand to be almost pulled out of bed by it. No evidence of hemianopia.

*On April 11*, the patient winked less when feinted at from the left than from the right side; he could not stand without support, the left leg was very weak, the foot somewhat inverted. He became steadily weaker and died April 14, 1907.

CASE 18. N. F. T., 42, salesman; admitted to Manhattan State Hospital January 4, 1905.

*Family History.* The father died of general paralysis, the mother of heart disease. Nothing was learned about the collaterals.

*Personal History.* The patient's mode of life had estranged his family so that a good history of his life could not be obtained. He was born in New York in 1860, received a good education, graduated at 24 from the university. He became a veterinary surgeon, but later he apparently followed a variety of occupations, was intemperate and dissipated. He married at the age of 28; two years later his wife died of puerperal fever, leaving him a daughter. He worked for several years as a salesman, and traveled for a medical book; he continued to lead a dissipated life and had delirium tremens in 1900. For some time previous to admission he had been "queer" and an annoyance to his relatives. He finally found his way into the workhouse, where he talked boastfully of his great wealth; he was later committed to Manhattan State Hospital.

*On admission* to Manhattan State Hospital (January 4, 1905), the patient was quiet and tractable, talked in a boastful manner about his wealth and abilities as a salesman; it was not possible to obtain from him any reliable information. His general health was satisfactory. He adjusted himself pleasantly to hospital life; during 1905 no special episodes were noted.

*Mental Status February 18, 1906.* The patient is quiet and good-natured, if not contradicted; he continually talks to the physicians, attendants and fellow-patients about his riches and his future plans. He is a millionaire, will give all the hospitals, churches and libraries $500,000; he intends to take all those suffering from rheumatism to Europe in a sumptuously furnished ship; he has been to the North Pole with Peary; the dead can be resuscitated by electricity (an idea borrowed from another patient). He talks incoherently, leaves his sentences unfinished, frequently sticks in the middle of a word and passes to another subject: "I was shot but Marcus four minutes was alive again electricity and rheumatism—the cemeteries are all empty—2,000 in Europe."

He has a fair idea of the date and place; his memory is very defective, he can give only a fragmentary account of his life with absurd inconsistencies.

His grasp of general information is much dilapidated. "9×7=63," "9×9=83—is 86," "9×7=18." Retention of tests is very poor. The patient has no insight into his mental impairment.

*Physical Status.* No external evidence of syphilis; he denies infection. Knee-jerks and Achilles-jerks equal, exaggerated; plantar flexion on both sides. Pupils slightly notched, irregular, react well on accommodation, but defectively to light. No marked weakness of either side, no sensory disorder. Gait uncertain, waddling, jerky; no sign of Romberg. Tremor of tongue, face and fingers. Ataxia in touching nose with index finger. Speech tremulous, sticking, with distortion of words; writing extremely tremulous with distortion of words. The patient has a slight cough; heart action is satisfactory; peripheral arteriosclerosis.

During the summer the patient became feeble and mentally more dilapidated.

*On September 1, 1906*, the patient had a series of convulsions lasting from 4 to 5 A. M.; these were localized on the left side of the body (attendant's note). When examined in the morning he lay with head and eyes turned somewhat to the right; the *left* face, arm and leg were in a state of flaccid paralysis; he appeared to be insensitive to pricks on the left arm and leg, but reacted to pricks on the left face. There was prompt reaction to pin-pricks on the right side. No sign of Babinski. He was very dull, did not speak, paid no attention to spoken gestures or commands. In the afternoon he regained the use of his limbs. On September 2 he was able to move the left arm and leg; the sense of pain on the left side was still impaired. He kept up a continual stream of incoherent talk. On September 8 he was able to be up.

*On September 18* the patient suddenly lost power in his *right hand*, was unable to articulate but smiled; the face was drawn to the left side. The speech difficulty and right-sided symptoms were noted next day; he did not use the right arm, the right leg was not so rigid as the left. He made no response to questions, did not obey commands, although he looked bright.

*On September 23*, he was able to give a good hand-grip with the right hand, and could talk; he reacted promptly to pin-pricks on both sides. He lay in bed repeating a senseless jargon or simple phrases.

*On October 4*, he was unable to take up a cup with the right hand; this persisted for a week. He was very feeble and on October 12 was placed in bed. He lay in bed babbling aimless remarks. There was extremely well-marked tremor of all extremities with coarse twitching.

He rapidly emaciated and developed a very deep bed-sore over the sacrum; broncho-pneumonia developed. The temperature had only occasionally reached 100° during October; on November 3 it reached 102°, then steadily sank to 97° on November 6. The patient died on November 9, 1906.

CASE 19. W. B., 45, salesman; congenitally left-handed, right-handed by education; admitted June 27, 1905.

*Family History.* Negative.

*Personal History.* The patient was born in Ireland in 1860. At the age of four he began to suffer from osteitis and went about in a wheeled cart until the age of ten. When he was a boy of six years he had to be broken off the habit of using his left hand; his left hand had to be tied at times. In later life he always used his right hand. In his youth his right patella was fractured. He was a bright scholar; on leaving school he entered the dry-goods business, and was an efficient salesman, and later a commercial traveler. He came to America at the age of 21 or 22. He was temperate in the use of alcohol. He had some venereal infection when a young man and probably had treatment for a very short time; there was no history of secondaries.

In 1903 the patient married; his wife did not become pregnant.

In *August, 1904*, occurred the first observed symptom. The patient one day sat down to write an invitation to an intimate friend; he *could not remember his name;* when the name was given him he *wrote it very badly* and recognized the fact, "look how I've written—I can't do it any better." No weakness of either side was noticed by his wife. About this time his speech was affected and his comrades occasionally thought (incorrectly) that he was drunk. He became very untidy at table, would spend hours looking at himself in the glass, and gave inadequate explanations, such as "I have to—I'm cutting my moustache." He was forgetful, absent-minded, made strange remarks, would repeat the same story several times. He complained of his feet, his gait became unsteady, he fell several times. In February, 1905, on hearing of the commitment of a friend he said "that's the way I'll go too." In the Spring of 1905 he was irritable, capricious, had little outbursts of passion; his conduct was more erratic, he was much more talkative than usual, neglected his personal appearance. From April until his commitment in June, he made life for his wife very disagreeable, he was pleased with nothing. In June, when his wife went to town, he refused to stay in the same house with her, stayed at an hotel. He now began to think that he had a lot of money, automobiles, horses, etc.; Tiffany owed him a million dollars. On one occasion he was unable to find his way home, and the night before he was taken to hospital he probably slept on the street. Two or three weeks before admission he had come in distress to his sister to borrow a quarter for breakfast, and on that occasion he said "I'm going all to the dogs."

*On admission* (June 27, 1905), the patient was quiet, accessible and good-natured, He answered questions relevantly, but made few spontaneous remarks; the date he gave correctly, but he did not know the name of the hospital. He said that he never felt better in his life, and uttered a variety of boasts about his present wealth and bright prospects. At the same time he had some insight into his sickness and made several references to this; he said that he had been broken down, that he had suffered much from rheumatism, and had much worry and bother. He looked forward to owning 150 horses and much land, lots of money, "automobiles, yachts, and everything that I desire." He gave a fair account of his life with no gross discrepancies, but was rather vague and incorrect in his statement of the manner in which he had spent the days immediately preceding admission.

Simple questions on general information frequently were too hard for him; he said "I have a very poor memory." Simple addition was performed correctly, but he was unable to subtract—"7 from 19 is 11."

Although admitting that his memory was poor and that he had some physical symptoms, he had no true appreciation of his general mental impairment.

*Physical Status.* The patient was a tall Irishman with no anatomical stigmata, but with several scars, some of which appeared to be of syphilitic origin; he admitted having had syphilis when he was young, but no trustworthy information as to symptoms and treatment could be obtained. The knee-jerks were absent; the pupils reacted well to light and on accommodation; some unsteadiness of gait and in the upright position was noted; there was tremor of tongue and fingers and the writing was very tremulous and untidy; speech was slurring and stumbling with omission of syllables. No other motor disorder was observed. He showed general diminution of the sense of pain. The internal organs were normal.

During the summer there was no marked change in the patient's condition and in November his physical status was practically the same; his gait at this time showed no disorder, he was able to walk steadily along a straight line. The pupils continued to react well.

Mentally he had deteriorated somewhat. His grandiose ideas were more dilapidated than on admission; he was very expansive and gave a farrago of grandiose ideas—he was the oldest man in the world, three years old, would get all the languages, possessed everything, was in the finest hotel—he introduced disagreeable features, such as "those nasty fellows there," "these are the worst rooms in the whole going to poison."

His stream of talk showed superficial associations, and frequently consisted of an incoherent enumeration of articles, especially of eatables, "Everybody is making all these things—I'm not making them—wines in Sicily and all these American towns—College Point, Constantinople and Boers and Boers and Boers and wild boars and horses and stallions and parasols for winter and summer."

The progress of the disease presented no special incident until November 21, 1905, when he complained at night that there was a man under the bed, who had come up through the floor and was annoying him.

*On November 22*, he was very weak, staggered and was put to bed; he shortly afterward was found lying on the floor and was unable to make himself understood. He showed marked *paraphasia;* he uttered with great effort, and evidence of much irritation, an unintelligible series of sounds. His *left arm* was quite limp and with the left hand he could only give a weak grip; the right hand-grip was strong. He did not show his teeth on request, merely pointed to the physician's teeth. When asked to write, he pushed the paper aside and wrote his name (William) on his left arm which was lying across his abdomen. He understood some simple questions. When examined a few hours later he showed marked paraphasia with perseveration, his utterances sometimes consisting in a mere jargon of syllables; *he could not write his name* but after starting correctly showed perseveration on one letter. When given a tumblerful of water he drank appropriately; when given eyeglasses and asked to indicate their use,

he took them awkwardly, showed no appreciation of how to put them on. Plantar flexion on both sides. He was rather excitable and irritable.

*On November 23*, the aphasic disorder was unchanged; the patient had considerable difficulty in swallowing, the milk would frequently trickle back from the lips. When assisted to walk he showed no noticeable weakness of the left leg, but occasionally he suddenly collapsed.

*November 24*, the patient swallowed liquids without difficulty. As on the previous day, he was unable to put a cup to his mouth; when a spoon was put in his hand he would grasp it correctly, but seemed unable to lift it. Tests for *hemianopia* made during the following week were inconclusive, but on November 25, he did not react to a moving object in the left field. The *paraphasia* persisted for several days.

*On November 26*, he had considerable difficulty in naming objects, he called a nickel "cellah—sevrah—silvah;" (how much?) "Fillabel—finv (?fünf)—its walless—its worth—eh;" he seemed to understand simple questions, but frequently made quite irrelevant or unintelligible replies, e. g., (Why are you in bed?) "be—because—too—cook me—B. (his name);" (Q. repeated) "because I didn't get what I was——(inarticulate)." He named colors correctly; in naming objects he frequently showed paraphasia and perseveration, e. g., (Paint brush) "Bush—for your teeth," (Shoe) "Toose,,' (What?) "Boo-b-bushes;" (Sock) "Sex," (Paper) "Bank-paper," (Watch) "Yatch—watch." He could not name objects placed in the left hand; he named correctly (with paraphasic fumbling) objects placed in the right hand.

No difference in the action of the facial muscles on the two sides could be made out. The *left hand-grip* was much weaker than the right, the left leg seemed weaker than the right (?poor co-operation).

Tests for hemianopia were not conclusive, but a moving object seemed to attract less attention on the *left* than on the right side.

*On November 27*, the patient was able to give as good a grip with the left hand as with the right, but showed difficulty in carrying out definite movements with his left hand. He was unable to pick a cent off the table with the left hand; when told to brush his nose with a little brush he had great difficulty in taking up the brush in his left hand, he then brought his hand to his nose with the brush in the palm; when further urged, he brushed his cheeks, not his nose. With the right hand he carried out the movement promptly and accurately. He had great difficulty in touching his nose with his left index; he did it easily with the right. The sensibility of the left hand to pin-pricks seemed the same as of the right. There was no evidence of the weakness of the left leg, which was observed on the previous day.

Tests for hemianopia were inconclusive. He still used paraphasic expressions.

*On November 29*, the paraphasia was still present; there was definite *astereognosis* of the left hand. He touched his nose promptly with the index finger of the right hand. When asked to do it with the left *index finger*, he touched his nose with his *hand* or made little gestures with the hand; when asked to touch it with the forefinger, he said, "I don't know what you mean." He finally did it correctly with the left hand and without any ataxia, but frequently he would wave the arm about before carrying out the movement. Tests of sensibility, carried out with a brush, were inconclusive; he would wave his arms about, rub one arm on the other, no matter which arm was touched.

In the evening he suddenly attacked the attendant, said that he would not let him kill his wife; he made frantic efforts to go out into the corridor; when allowed to go in order to investigate, he hurried to the opposite side, hunted under the tables; he struggled violently when taken back to bed; he pointed at the attendant, said, "there's my wife."

*On December 1* the patient was still rather excited, frequently struggled to leave the bed; he talked quickly, stuttered badly, was frequently quite inarticulate. He named many objects correctly, but with others he showed paraphasia. (What is this? 5c.) "That's a V—omen—Victoria—that's villeela—et—what—Victoria is it—vill—who? me—me—v—u it's done—I didn't say it was Victoria at all—well if I didn't they wouldn't give you that."

Tests for astereognosis and hemianopia were quite inconclusive. He gave a weaker grip with the *left* hand than with the right. In the evening he appeared to be following with his eyes objects on the wall to his left.

*On December 2* he was rather dull and difficult to rouse; he made no reaction to severe pin-pricks on either side, but referred to "the feeling—just where you stab me." He moved both hands equally freely. No difference in reaction to objects in the left and right visual fields.

*On December 5* the patient was much brighter and practically in the condition preceding the attack; he could now pick up a cent as easily with the left as with the right hand. His writing was slightly worse than on admission to the hospital.

The whole episode, therefore, had lasted rather less than two weeks.

During the next seven months little change was noticed in his general condition, but several times he answered imaginary voices, and said that he actually did hear voices; he would pound the chair in his rage and swear vigorously, but, when asked about his experiences, he would look ashamed and refuse to discuss the matter. He fell on one occasion and sustained a fracture of the neck of the femur, which did not unite.

*On July 17, 1906*, after a bath, his eyes and head turned to the left,

the eyes twitched, he could not speak although he appeared to make an effort. At 10.05 A. M. he had a general convulsion. When examined at 10.35 he was lying on his back with head and eyes turned to the left, the *left angle of mouth* puffed out during expiration; *limpness of left arm and leg;* he could give a right-hand grip, but not a left. Sign of Babinski on the left; on the right side the plantar reflex was sometimes flexion, sometimes extension. He did not react at all to pin-pricks. In the course of the day he had three general convulsions. When examined at 5 P. M. there was evidence of *left-sided hemianopia;* he paid no attention to objects in the left field, but followed objects in the right field and grasped at them; feinting from the left side caused no reaction, from the right side caused winking. He made faint inarticulate sounds when addressed, he made at first no attempt to name objects shown, but later called a pencil "stencil—pencil." During examination the head and eyes turned to the extreme left, the eyes, forehead and facial muscles twitched; this passed off in a minute. He reacted to pin-pricks on either side of the face, not on the arms nor body. A little later the left arm gave three or four clonic contractions, the left leg began to jerk, then a general convulsion developed; the head and eyes were pulled to the right. The left arm was the first limb to relax. At 8 P. M. there was evidence of *left-sided hemianopia;* objects in the left field were not reacted to, in the right field were followed by the eyes and grasped; feinting at the eye from the left side never elicited a reaction; from the right side frequently caused winking. No reaction to pin-pricks even over the face. He could not be brought to grip with the left hand, gripped well with the right; left leg more flaccid than the right; sign of Babinski on both sides.

During the examination the head and eyes were suddenly turned to the left, the left arm began to twitch, the mouth was pulled to the extreme left; the left leg now began to twitch and the convulsion then became general; after a few seconds of stertorous breathing with cyanosis the head and eyes turned to the right.

The patient had another convulsion during the night and next day was in a comatose state. On July 19, at 9 A. M., he died.