

function is to visit all the relatives of the patient, the family physician, and any other person who can give information about any member of the family as far back as it can be traced.

An attempt is made to obtain a detailed life-history of every such member of the family, including a description of the mental and physical state, habits, illnesses, and cause of death.

The "field worker" is armed with all previously acquired information and interviews the patient just before visiting the friends. She is given explicit instructions as to the particulars to be elicited, but uses no printed form in noting them. Where relatives exist in localities worked by other investigators she is expected to record their addresses for the use of the latter. Several visits are generally found necessary to complete the pedigree. The "field worker" is also found useful as a means of keeping in touch with discharged patients and of discovering other individuals who require care. Every attempt is made to establish a friendly footing with the patients' relatives and apparently with success in America. The investigator is instructed in the Mendelian rules of heredity that this understanding may direct inquiries, but she is warned against being biased in the collection of facts by any knowledge of what is expected. The paper includes a brief statement of the Mendelian rules for the transmission of simple unit characters.

The method of recording data which is described includes a mode of charting recommended by a committee of the American Association for the Study of the Feeble-minded. It is one which enables the clear expression by means of symbols of practically all the points of importance about every discoverable member of the family.

The second of the papers mentioned above is an example of the application of these methods to the study of the transmission of feeble-mindedness. It includes fifteen pedigrees of patients admitted to the training school at Vineland, N.Z. Each pedigree records the presence or absence of feeble-mindedness and allied traits in every member of all branches of the family for three, four, or even five generations.

Such pedigrees naturally involve an enormous amount of labour in collection, and this is only a preliminary report. No statement is made as to the standard adopted in labelling individuals as feeble-minded. The pedigrees are presented without analysis to determine how far the transmission accords with Mendelian rules. In general they suggest that the defect behaves as a recessive to the normal state. To the reviewer, however, it appears that the mode of transmission in a few instances is incompatible with this simple formula. In order to include others within it it would be necessary to make assumptions for which there is no evidence without the history of a generation either preceding or succeeding those actually investigated.

EDWARD MAPOTHER.

### 3. Clinical Psychiatry.

*Amnesia in General Paralysis [L'Amnesia dans la Paralyse Generale].*  
(*Gaz. des Hôp.*, Aug. 5th.) Benon, M. R.

This paper consists of a discussion of the fundamental characters of

the amnesia of general paralysis of a special character according to the stage and type of the disease, and of its diagnostic significance.

In the first place, the fundamental characters of the amnesia in this disease are stated as follows: It is both anterograde and retrograde, *i.e.*, the defect is seen in regard to events both subsequent and prior to the onset of the disease. All the functions of memory are affected. There is weakening of the capacity to register fresh impressions and instantaneous amnesia may be seen. The process of revival of memories is retarded or lost. The power of attributing to recollected events their proper setting in time and place is also impaired. The enfeeblement of memory is diffuse, though not necessarily profound in the early stages. It shows no tendency to systematisation. The amnesia is not exclusively for the events of a certain period of the patient's life, nor for a series of associated events. It does not especially effect acquirements of a certain area, *e.g.*, professional or scholastic, nor is there any evidence that memories of one type, such as visual or auditory, are picked out. Further, the enfeeblement is progressive. A striking feature is the indifference of the patient to his own amnesia, and such indifference is very suggestive of general paralysis.

In the second place, the author describes at length the characters of the amnesia at different periods in the evolution of the disease.

It is, as a rule, one of the earliest signs, and during its onset amnesia having the characters described can generally be found. The forgetfulness may not be very obvious and may fail to attract the notice of unobservant relatives. It generally first becomes noticeable in business relations, especially where the occupation is an intellectual one. The author believes that much of the eccentricity of conduct in the early stages of general paralysis is referable to amnesia, though defect of judgment and emotional indifference are accessory factors. In this stage there may also occur attacks of definite dissociation. Such attacks generally last a few hours or days but may be prolonged even for months.

As the disease progresses one observes an increase in the number and simplicity of the facts which the patient has forgotten. When questioned he answers quickly and at random. If he gives an occasional correct answer, it is impossible to get him to elaborate his statement. The date of birth and name of birthplace are usually among the last facts to be forgotten.

In the terminal stage, memory may be said to have disappeared. The patient barely recognises his most intimate acquaintances. He fails to understand the meaning of questions.

During remissions, the amnesia of the general paralytic becomes much less obvious, but does not lose its essential characters. Lapses of memory still occur; they do not worry the patient in the least. Moreover, if one reminds him of misdemeanours committed during his period of more marked impairment, he either denies them vigorously or exhibits extreme indifference. The author concludes that complete remission of mental symptoms does not occur.

Amnesia having the general characters described above is to be found in all types of general paralysis. Where melancholia, excitement, exalted delusions, or seizures (either epileptiform or apoplectiform)

constitute the dominant feature of the clinical picture, this amnesia may be determined, and it serves to distinguish general paralysis from the numerous conditions with which it may be confused. It is particularly useful in serving to discriminate from general paralysis other psychoses associated with tabes.

The amnesia of localised organic lesions (*e.g.*, syphilitic) is mainly distinguished from that of general paralysis by the attitude of the patient towards his own defect. He is generally unaware of its existence until his attention is called to it—though this is not always the case as in general paralysis. But then one finds that he is intensely worried by his defect, struggles to recollect, concentrating his attention upon a question and evincing obvious satisfaction or depression according to his success or failure. He also frequently takes precautions to obviate the results of his forgetfulness after recognising it.

The amnesia in such conditions is also frequently lacunar rather than general, and is often less marked after mental rest.

The author concludes that the amnesia of general paralysis presents in itself nothing absolutely pathognomonic, but that with the disorders of judgment and of emotion it constitutes a mental state which is of greater diagnostic significance than physical signs in difficult cases.

EDWARD MAPOTHER.

*A Case of Tubercular General Paralysis [Un cas de paralysie générale tuberculeuse]. (Bull. Soc. Clin. Méd. Ment., Dec., 1910.) Pactet et Vigouroux.*

This paper records the pathological findings in a case previously exhibited before the Clinical Society. It was that of a youth, *æt.* 20, who had suffered from a typical progressive paralysis since the age of fourteen. He was demented, with unequal pupils, the light reflex being abolished, increased knee-jerks, hesitant speech and uncertain gait. Lumbar puncture revealed no lymphocytosis, and at no time had he any seizures. There was no history of syphilis either in the patient or his parents. He had, however, a tubercular family history, and had suffered from suppurating tubercular glands in legs, neck, etc. His death was due to acute pulmonary tuberculosis.

His brain showed the degenerative rather than the inflammatory changes found in general paralysis. There were no local brain lesions. The membranes were thickened and milky, but not adherent. There were no granulations of the ventricular ependyma. The vessels were little affected, but the perivascular spaces were dilated, and contained albuminous fluid with few cells. There was some hyaline degeneration of the smaller vessels. The cells were much altered in all parts of the brain. The chief alteration was a pigmentary degeneration. The neuroglia was proliferated, and spider-cells were numerous.

The authors look on this as a typical case of the variety of general paralysis due to tubercle, as differentiated by Klippel. The paper is illustrated by two plates.

W. STARKEY.