Tabes in asylum and hospital practice / by F.W. Mott.

Tales —

by

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TABES IN ASYLUM AND HOSPITAL PRACTICE.

BY F. W. MOTT, M.D., F.R.S.

Introduction.

As I have pointed out in an article on "Brain Syphilis," an artificial classification of diseases obtains owing to our system of administration of relief to the suffering poor, by which asylum doctors see particularly those cases with mental symptoms, while hospital physicians meet with those cases especially which manifest physical signs of paralysis, sensory disturbances, &c. Thus it is with tabes and general paralysis. But in the almost unique position in England which I hold, of being able to study diseases *both* in hospital and asylum practice, and with the vast field which the latter offers to me as Pathologist to the London County Asylums, I am enabled to study all types of this disease from cases presenting purely spinal symptoms, to cases presenting mental symptoms which would lead to a diagnosis of general paralysis.

In order to increase my field of observation, and also with the knowledge that some of the cases might eventually come under further observation at one or other of

the various asylums, I have visited a number of the infirmaries in order to study cases of tabes dorsalis in the paralytic stage or with premonitory mental symptoms. Some of these cases have since come into the asylums and died; others, I have found, had relatives, consanguineous or conjugal, dying in the asylums. I have been enabled to obtain thirty *post-mortem* examinations, and a systematic macroscopic and (in the great majority of instances) microscopic examination of the brain and spinal cord has been made of each; frequently, also, the spinal ganglia, peripheral nerves, optic nerves, retinæ, and other structures, have been examined.

In some of these cases a full and detailed report will be given; but where it is only wearisome repetition, this will not be done; but the facts will, I trust, be accepted as true, being deductions from my personal observations on many thousands of sections of the central and peripheral nervous systems. Likewise with the clinical notes of the cases, which include more than sixty cases of tabes dorsalis and sixty cases of the tabetic form of general paralysis, I shall only record in full those cases which present some very interesting facts or rare condition.

I have examined systematically the sensory disturbances in fifty cases of tabes dorsalis, and in a number of such cases of tabetic general paralysis which were capable of giving reliable answers; also other clinical signs and symptoms, including the mental condition. The amount of work involved in this can only be appreciated by those who have had a similar experience, and, of course, this research is not yet ended, for the majority of the cases I have observed clinically have not yet come to the *post-mortem* table. I found it advisable, owing to the vast amount of material at my disposal, to limit my observations to one class of disease, and in selecting this, tabes dorsalis, I thought it was possible to associate some of the mental disturbances with the changes which one meets in the nervous system, and to show that the illusions, delusions, and hallucinations have an organic basis, and that there is a great analogy between the paroxysms of pain, the visceral crises of tabes,

and the epileptiform fits, attacks of mania, delirium, hallucinations, and other mental disturbances which occur in general paralysis.

In the study of these cases, just as one can trace all grades of organic lesions from the purely spinal, through spinal with slight cerebral change, to extreme cerebral change and slight spinal change, so one can trace corresponding clinical symptoms. Many cases begin with marked spinal change and accompanying spinal symptoms; there is then a sudden or gradual outburst of mental symptoms and the spinal sink into the background. So much may the cerebral symptoms overshadow, obscure, and even obliterate the spinal symptoms, that they may be overlooked and disregarded. In fact, I have seen so many cases in which ataxy has disappeared with the progressive cerebral disease that I am reminded of the truth of the dictum of Hughlings Jackson that "one half of the symptoms of nervous diseases are due to the unbalanced action of healthy structures." That many of these cases were in the second stage of tabes, and yet evinced after the onset of the mental symptoms little or no ataxy in their movements, was shown by the examination of their spinal cords and posterior roots. At a discussion opened by myself at the Pathological Society upon the unity of tabes and general paralysis, most of the leading authorities—Sir William Gowers, Dr. Buzzard, Dr. Savage, Dr. Ferrier, Dr. Hale White, Dr. Head, and others, who spoke—were of the opinion that etiologically and pathogenetically the two diseases were identical. This view has not, however, found its way into the text-books, although, as a matter of expediency, it is often adopted by physicians, for when a noble or distinguished patient suffers from grandiose delusions and other signs of the progressive brain disease which in a few years will terminate fatally, it is given out that he is suffering from locomotor ataxy. I maintain that etiologically and pathogenetically there is *one tabes* which may begin in the brain (especially in certain regions), or in the spinal cord in certain regions, or in the peripheral nervous structures connected with vision, or in nervous structures con-

nected with the viscera, constituting, therefore, different types, any of which may be present or be associated with one or all of the others. More and more we are coming to the opinion that syphilis is the cause of the degenerative process evinced, and although many authorities will not go so far as Möbius, who considers that all cases are meta-syphilitic, or Fournier, who calls them para-syphilitic, still the majority of neurologists and alienists in England and Germany believe that syphilis is the main causal factor of this polymorphic disease. The experimental observations of Krafft-Ebing, and the fact that practically all cases of hereditary tabes and almost all cases of juvenile general paralysis can be proved to have occurred in the children of syphilitic parents strongly supports the syphilitic doctrine of this disease.

Current Views of Relation of Tabes to General Paralysis in Standard Text-Books.

Since Westphal showed the existence of posterior-column-sclerosis in cases of general paralysis, nearly all neurologists have gradually come to recognise a close alliance between tabes dorsalis and general paralysis. It would be impossible to relate all the literature that has been written on this subject, but I will give a few quotations from some leading alienists and neurologists. The first in this country to recognise the association was Dr. Savage, and he says (Article on Locomotor Ataxy, "Tuke's Dictionary of Psychological Medicine"):

"Locomotor ataxy and insanity may occur in the same person and be unconnected, or locomotor ataxy may precede the development of associated mental symptoms, or locomotor ataxy symptoms may be the first indication of general paralysis of the insane. Locomotor ataxy and general paralysis may to some extent alternate, so that while the ataxic symptoms are fully developed the mind is clear; and while the mind is disordered the ataxy becomes less or is absent. Locomotor ataxy may have the following special reactions mentally: there may be during the course of the disease mental crises; a patient who is recognised as suffering from locomotor ataxy suddenly becomes manaical.

In these cases generally there is more or less suspicion, or a tendency to retaliate on those who are supposed to be causing the painful sensations in various parts of the body. These maniacal attacks are of short duration, but may recur at regular intervals. There may be insane interpretations of the ordinary crises, so that one patient says that his bowels have been twisted by his persecutors, and another says that red-hot irons have been thrust in his feet and eyes, and another complains that unnatural means have been used to withdraw his semen. The ordinary symptoms of locomotor ataxy are insanely explained in other ways. The mental symptoms of these cases may be acute or chronic; in the former case they may alternate, so while the delusions exist the ataxy is better, and *vice versâ;* or the insanity may be transient or recurrent. In some cases the insanity may be as chronic as the locomotor ataxy, but there seems to be little tendency to dementia in these patients. The most common relationship of insanity and locomotor ataxy is met with in general paralysis of the insane, and in this the symptoms of both may begin at the same time, so that with extravagance, boastfulness, and lust ataxic weakness may develop. In other cases locomotor ataxy is the first symptom, and after a period varying from one to seven years, other symptoms point to the existence of general paralysis. In some cases the general paralysis has first been recognised, and it is only later that locomotor ataxy is recognised."

Mickle (Article on General Paralysis: "Tuke's Dictionary of Psychological Medicine") merely states that "the ascending form of general paralysis must be distinguished from tabes dorsalis, at times a difficult task," otherwise hardly any notice is taken of this form of general paralysis.

Bevan Lewis ("A Text-Book of Mental Diseases; Pathological Anatomy of General Paralysis," p. 566). Referring to spinal symptoms, this author groups cases into four arbitrary divisions. I will content myself with quoting in full his remarks in regard to the second group, comprising from the very onset notable tabetic symptoms:—"The cerebral symptoms are often so greatly in abeyance as to arouse the doubt whether we are not here engaged with a genuine tabes dorsalis of spinal origin. The disturbance of sensation, the abolition of the deep reflexes, the ataxic gait, are all so prominent that we are apt to attribute such symptoms to a primary implication of the cord itself. (This, I am sure, is the actual cause of the symptoms, and my own observations show that we should be right in thus accounting for them. F. W. M.) Yet in this tabetic form of general paralysis we must usually witness complete subsidence of the special spinal symptoms, the

tabetic gait passes off, the knee-jerk returns, and then the full development of the cerebral symptom is established, or what is not infrequent, the sensory implication of the cord becomes a motory affection, and the spastic paraplegia replaces the anæsthesia and ataxia. (My observation is that these clinical facts are right, and the inference wrong. F. W. M.) There has been a tendency to regard the later evolved cerebral derangements of typical general paralysis established in a well-marked tabetic case as due to an ascending change—that is, propagation by direct continuity of diseased tissue, thus making a system disease of the spinal cord the originating factor of the subsequent 'lesions of general paralysis.'" Dr. Lewis would look for the explanation rather through the vaso-motor agency operating on nervous systems in physiological sympathy with their higher centres. I will discuss this point later, but with the first statement, that it is not an ascending change, I quite agree.

On page 319 Dr. Lewis states: "In tabetic forms of general paralysis the oculo-motor anomalies almost invariably precede the tabetic signs of abolished knee-jerk, and the pupils failing to dilate on shading or cutaneous stimulation, a paralytic myosis (moderate) eventually passes into a genuine spastic myosis from the irritation of a diseased process advancing upon the constrictor nucleus."

On page 322: "We may enumerate the association of a paretic or tabetic gait with the abolition of the deep reflexes and like irido-motor troubles. The tabetic gait occasionally associated with this absence of the knee-jerk is occasionally peculiarly disorderly, hurried, spasmodic, and insecure."

Régie ("Manuel Pratique de Médécine Mentale") says: "Since attention has been drawn to the mental state of tabetics, psychic troubles have been recognised more frequently in them; they are generally disturbances of intelligence and modifications of character which are revealed by irritability, bad temper, moroseness, hypochondriasis, depression and suicidal tendencies; at other times this may happen in the pre-ataxic stage; sensory troubles may be manifested, consisting of illusions, or more or less conscious hallucinations, principally affecting vision, hearing, or general sensibility; but the psychic disorders may not terminate here, and in certain cases insanity may co-exist. Pierret et Rougier, who have made a practical study of this insanity, have shown that more often it was a state of melancholia, with vague ideas of persecution and confused hallucinations. (A number of cases of this kind in my experience will be given later on. F. W. M.) The patients accused persons of wishing to poison

them, of making them burn; they complain of hearing that injuries will be done to them, of experiencing a bad taste in their food and in their mouth, of experiencing electricity in their limbs and disagreeable sensations; the delirium may present itself in the hypochondriacal form or in the ambitious form, as in general paralysis. Finally, tabes may be accompanied by enfeeblement of intellect, which may present great difficulties in distinguishing it from general paralysis. In some cases the disease assumes a hybrid form, and seems to be both tabes and general paralysis. The latter may be primary or consecutive to locomotor ataxy. When it begins thus by spinal phenomena it is given the name of ascending general paralysis. (This, I hold, is wrong. F. W. M.) This is the place to repeat that general paralysis and tabes are diseases absolutely similar as regards origin and nature, and that they have the closest relation to one another. It is not rare to see general paralysis begin or end by symptoms of ataxy, and in certain cases present during the course of the disease a mixture of spinal and cerebral symptoms."

Krafft-Ebing ("Progressive allgemeine Paralysie, Nothnagel's System"), one of the most energetic supporters of the syphilitic doctrine of general paralysis, strangely enough does not devote much attention to the relation of this disease to tabes dorsalis. On page 11 he refers, under motor disturbances, to tabetic symptoms in the form of temporary ocular paralyses, myosis and reflex pupil rigidity in the prodromal stage. On page 21 the disease of the spinal cord appears as "atrophy of nerve-fibres with consecutive development of connective tissue, and it may be looked upon as corresponding with the tabetic process. It is most strongly developed in Goll's column, and in the cervical cord is mostly limited to this tract. (Westphal.)"

On page 49: "The motor defects of the trunk and extremities are different in nature, and partly due to muscle insufficiency, partly ataxy, partly tremor. They arise in part from cortical changes, partly from pre-existing and complicating changes in spinal tracts. The gait may be paralytic, spastic, or ataxic."

On page 54: "In about 4 per cent. of the cases of paralysis, one finds tabic optic atrophy."

Page 73: "Often spinal cord changes, such as diseases of the posterior columns arising from the same predisposing cause—syphilis, whereby simultaneously or successively spinal cord or brain are affected. The tabetic process may come on with the paralytic or precede it." (He does not, however, mention it in the differential diagnosis. F. W. M.)

Sir William Gowers ("Diseases of the Nervous System," Spinal Cord, p. 466) says :

"Another very important and frequent complication of tabes is general paralysis of the insane. The two diseases have many alliances; it is probable that syphilis is the chief cause of general paralysis as well as of tabes. Reflex irido-plegia is common in both diseases; the two maladies are often combined, and the symptoms of one or the other may preponderate. Thus many general paralytics present symptoms of tabes, and its characteristic lesion is found after death. On the other hand cases of tabes may present slight symptoms of general paralysis, perhaps only slight optimism and mental weakness, which may remain subordinate or may increase to a pronounced or preponderate degree; it may be difficult to say in which category a case should be placed. It is sometimes said that a disease may commence as ataxy and change to general paralysis, but a more correct expression of the facts is a co-existence of the two affections and the dominance of the symptoms of one or the other."

Clouston ("Mental Diseases," 5th Edition, 1898, p. 390) considers :—

"The most marked variety of general paralysis is the peripheral form, where the pathological process does not begin in the cortex of the brain but in the cord (the tabic form), or in the neuronic portions of the organ of special sense (the sensory form), or in a peripheral nerve (the peripheral form) spreading upwards by pathological propagation along the connecting nerves in the lines of physiological function till it reaches the brain cortex. These varieties are rare, but distinct enough when they occur, and very interesting. They would seem to imply that the pathological process of general paralysis resembles the progressive Wallerian degeneration."

This assumption of Prof. Clouston is purely hypothetical; I have never seen degeneration of the fillet in the twenty-five cases of the tabic form of general paralysis which I have examined microscopically. If there were a progressive degeneration spreading up to the cortex along the path taken by the kinæsthetic impulses, there would be a continuation of the degeneration of the column of Goll, which was invariably present in all these cases, through the posterior column nucleus, the internal arciform fibres and the fillet; but this I have never seen. Neither have I seen any evidence in support of a process spreading up from the peripheral nerves; no doubt Dr. Clouston has put in a hypothetical way the so-called ascending form of general paralysis. This author refers to six cases of the typical tabic form which he has seen;

strangely enough in an otherwise admirable text-book he almost disregards the influence of syphilis as a causative factor in the production of general paralysis. He refers to the opinion of Dr. Drummond, of Newcastle, but surely the work of Fournier, and a large number of other Continental authors who have worked at this subject should not have been disregarded ; and whatever Dr. Clouston's own opinion on this subject may be, in a text-book the other side of the question should have been stated, seeing that it is a prevailing opinion in England, America, and on the Continent, that, if syphilis be not the sole factor, it is the most important factor.

Möbius (" Ueber die Tabes," Berlin, 1897), in one of the most valuable monographs that has appeared on the subject, on page 6 of the introduction, points out that Türck, Baillarger, Simon, Westphal, Falret, Magnan, and others have studied both the clinical as well as the anatomical relations of the two diseases. However, it is only recently that the opinion has been advanced that really both diseases are of the same sort ; that we call it tabes when especially the centripetal nerve-fibres are diseased, and general paralysis when especially the cerebral cortex is affected.

" This view is by no means accepted by all, still I maintain it is correct. Both diseases have the same cause, for both are meta-syphilis ; in both there occurs a primary atrophy of nervous structures. Important symptoms, especially reflex pupil-rigidity and ocular muscular paralysis, are in both nearly equally frequent ; in many tabetic cases, slight changes appear to be present in the cerebral cortex, which essentially resemble those of general paralysis. Still more frequently are spinal changes, loss of knee-jerks, disease of the posterior columns present in paralytic patients. The tabetic cases come under the neurologists, and the paralytic cases under the alienists."

On page 31 on " The Brain Symptoms of Tabes," Möbius says :—" Attacks of migraine, epileptic attacks, apoplectiform attacks, and psychical disturbances may occur ; it is, however, more correct to consider these complications as symptoms of progressive paralysis in tabetic patients. They represent an incomplete paralytic disease of the cerebral cortex, and they correspond to the transitions between the cases with isolated paralytic symptoms and those with undoubted progressive paralysis."

Marie (" Lectures on the Spinal Cord," *Sydenham Society Trans.*, p. 292) says :

" The psychical derangements which occur in the course of tabes are not infrequent, but vary considerably ; and Dieulafoy

was able to apply the term 'Tabid Insanity' to the condition which existed in some patients, while others present similar symptoms which are but slightly pronounced, and may be either transitory or permanent, or possibly occur in paroxysms (Fournier). He distinguishes the moral from the intellectual derangements—the former he attributes more especially to the weak condition of the patient, and he remarks that notwithstanding the agonising pains the patients frequently feel, they are rarely found to commit suicide; while, on the other hand, in certain affections, notably those of the bladder, suicide is relatively more common. This is an interesting point in connection with the psychology of the tabic patient. (Both these symptoms are among the most frequent in tabes, and I have recollections of several patients who have attempted suicide on account of the pains and the miserable depression occasioned by an incurable disease, and accordingly been admitted to the asylum. F. W. M.) As regards the intellectual disorders, they are far less frequent, and if truly pronounced, depend upon the association of cerebral lesions with those of the spinal cord. Tabes is in that case most often found to be complicated by general paralysis of the insane. Such cases are rare, if not exceptional."

Etiology of Tabes and Tabo-Paralysis.

In the first volume of the Archives I gave a pretty full account of the literature in reference to the relation of syphilis and general paralysis both in the adult and juvenile form; since then, the percentage of syphilitic antecedents in general paralysis has attracted more attention in most of the London County Asylums, and I notice in the report of 1901 from Bexley Asylum that Dr. Stansfield gives 80 per cent. of the general paralytics with signs or history of syphilis. Dr. Thomas, of Hanwell, informs me that amongst the male admissions at Hanwell during the past eighteen months, 80 per cent. gave a syphilitic history. Dr. Bolton's statistics give similar results.

In 1863 Eisenmann and Topinard both expressed the opinion that syphilis was probably the cause of tabes; Westphal (1881), on the other hand, not only said that it was not proved, but it was not probable that syphilis was the cause of tabes. It was not until Fournier in 1876 brought

forward a series of thirty cases of tabes in which syphilitic antecedents were proved in twenty-four, that really attention was directed to the subject. As in general paralysis, so in tabes it was received with scepticism by the majority of the profession. Vulpian supported Fournier, and the latter collected more cases and in 1882 brought forward 103 cases of tabes, ninety-nine of which had suffered with syphilis, that is, over 90 per cent. Later he increased the numbers to 146, or 93 per cent. Erb in Germany, and Sir William Gowers in England collected cases, but it is especially the work of the former that has substantiated the doctrine of Fournier. Erb commenced collecting cases in 1879 and in the year 1897 his cases amounted to 900 of which 90 per cent. had suffered with syphilis. He found in 6,000 other nervous cases on the same basis only 20 per cent.

Westphal, Charcot and Virchow were always opponents of the doctrine, but the greatest opponent has been Leyden. Various authors have given different results. Very much depends on what each considers sufficient evidence to warrant the assumption of syphilitic antecedents. In a certain number of cases there is a history of a soft sore ; in a large number it would be perfectly legitimate to consider this was syphilis, even if the secondary symptoms were so mild as not to have been noticed by the patient. In women, still-births and miscarriages or sterility would indicate probable, but not certain, syphilis.

Statistics Relating to Syphilitic Antecedents by Various Authors, after Redlich.

Buzzard	45 per cent.	Séguin	72 per cent.
Fränkel	50·7 ,,	Collins	75 ,,
Gerhardt	51 ,,	Friedheim	75 ,,
Bernhardt	60 ,,	Voigt	76 ,,
Eisenlohr	60 ,,	Rumpf	85 ,,
Mayer	60 ,,	Althaus	90 ,,
Borgherini	61 ,,	Raymond	90 ,,
Remak	63·5 ,,	Strumpell	90 ,,
Gowers	70 ,,	Dejerine	97 ,,
Mendel	70 ,,	Quinquadel	100 ,,
Senator	70 ,,		

There is a very considerable difference in the statistics of men and women as regards syphilis. Erb's statistics (1896) are in women 57 per cent. certain syphilis, and 30 per cent. highly probable. Minor published eight cases of tabes in women in all of whom syphilis was present. Redlich's are 23·4 per cent.

Max Nonne points out that as interest in this question was increased the authorities who had previously only found a small percentage of tabetic patients suffering with syphilis were led, as the enquiry progressed, to increase their percentages; thus Berger's statistics rose from 20 to 43 per cent., those of Bernhardt from 21 to 60 per cent., Oppenheim's from 17 to about 80 per cent., and Rumpf's 66 to 80 per cent. Minor's observations relating to tabes are also of great interest. He showed that among 4,700 non-Jewish Russians 2·9 per cent. were tabetics, on the other hand among 698 Jewish patients suffering with nervous diseases only 0.8 per cent. were tabetics. Functional nervous diseases are extremely common amongst the Jews, and yet tabes is comparatively rare. This may be explained by the fact that the Jews are much less liable to become infected with syphilis. Minor later showed that both tabes dorsalis and syphilis are five times as common among the non-Jewish Russians as in the Jews of Russia. (Among my cases there were three tabetic Jews. Two certainly had had syphilis and one probably. There were also three tabic paralytic Jews all of whom had signs and history of syphilis.)

Statistics of Syphilis in Sixty-two Cases of Tabes Dorsalis.

In my own statistics there were forty-seven males and fifteen females. Of the forty-seven males, thirty-three (over 70 per cent.) had certainly syphilis, fourteen were doubtful, three owned to gonorrhœa and two to soft sore; nine denied infection, but only one of these was able to say that he had not been in the way of getting it, and this man very possibly may have been the subject of inherited syphilis or have acquired it in some unusual way when young (*vide*

Case 1). In connection with this point I may mention that I had a patient not long ago under my care suffering with epileptic fits which were cured by mercury and iodide. The patient was aged 20 and I was informed that he had contracted syphilis when a little boy by being allowed to sleep with a shop assistant who suffered with the disease. Again, children may present no signs of syphilis on the body, and yet when a careful history is obtained, congenital syphilis can be proved to be the cause of a primary optic atrophy (*vide* Case 74), juvenile general paralysis, or tabes.

Again, as an example of syphilis acquired in an unusual manner, I will cite a series of ten women infected in childhood by a midwife (*Lancet*, p. 402, 1895). Also, a number of glass blowers at St. Helens suffered with chancre of the lip. Many people are infected and never know they have suffered with the disease; but a larger number know and will not own up.

Statistics of Syphilis in Sixty-two Asylum Cases of Tabo-Paralysis.

Of fifty-four male tabo-paralytics and insane tabetics, forty-six of which were personally seen and examined by me, there was a reliable history, or signs on the body, of syphilis in at least 75 per cent. In four cases there were no notes obtainable, and I did not see the cases during life. In four there were doubtful signs or history. In only one case was there no history of syphilis, no signs on the body, and a healthy family, but as everyone knows, this does *not absolutely* exclude a syphilitic infection. In the remainder syphilis was probable, or could not be excluded.

There were eight women. In only two were there definite signs on the body; the history, however, of the remaining six made it almost certain that they had been infected. One was probably a "puella publica" without friends. Another was a kept woman who had previously suffered with venereal disease, and had had several miscarriages. The remaining four had husbands who suffered with tabes or general paralysis themselves (*vide* pp. 18, 19).

Birth and Death Rate of Tabic and Tabo-Paralytic Patients.

Twenty-six tabetic men were married, and had eighty-six living children, forty-seven dead, twelve born dead, and twenty premature births. Several were infected after marriage, and then followed miscarriages and dead children. It will be of interest to contrast the death rate of the children of ataxic patients of the two sexes.

Amongst the fifteen females, fourteen were married women. Four of these were sterile, and had no premature births; all gave a definite history or signs of syphilis on the body. Of the others one had a living child after ten years of married life; she did not know that she had had any miscarriages, and there were no signs of syphilis on the body. Of the remaining nine, three had certain signs and history of syphilis, but all the nine had miscarriages, still-born and dead children. One woman was unmarried, and there was no history or sign of syphilis. The record of the fourteen married women as regards children was as follows: Four had no children and no miscarriages; ten had amongst them (each contributing, so that every one was capable of conception and of child-bearing) six living children, sixteen miscarriages, fourteen born dead, and four dead in infancy.

The eight females had amongst them sixteen miscarriages or premature births, eight children dying in infancy, and ten children living, but the eight children dying in infancy, and five of the living children were contributed by one woman.

The married male tabo-paralytics were thirty in number, and had sixty-eight children alive, twenty-six children dead, and twenty-seven miscarriages or born dead. If we contrast these statistics of tabo-paralytics with the tabetic cases we find that they have fewer healthy and dead children. This is because they do not live so long after infection as the tabetics, moreover after removal to an asylum no further conception takes place, otherwise there is the same close similarity between the hospital and asylum cases. Adding the fifty-four males married, suffering with tabes or tabo-

paralysis together, and contrasting them with the twenty-two married females, we find a remarkable contrast, showing that when the female is infected, the chance of living children being born is greatly reduced; correspondingly, there is an increase of dead children and miscarriages.

	Children alive.	Born alive, but died in infancy, or afterwards.	Born dead.	Miscarriages.
Twenty-two married females, suffering with tabes or tabo-paralysis. Seven of these were sterile.	10	10	18	31
			49	
Fifty-four married males, suffering with tabes or tabo-paralysis.	151	75	52	

Several male cases were of interest because of a definite history of infection occurring after marriage: in these cases, miscarriages and abortions followed healthy children. It will be seen that very nearly 30 per cent. of the married women were sterile. Mendel[1] found that in 252 married female tabetics 32·9 per cent. were childless.

Statistics Relating to General Paralysis in Women.

By kind permission of Dr. Stansfield I am enabled to publish the following data from statistics furnished by Dr. Hubert Bond:—

[1] E. Mendel. "Die Tabes beim Weiblichen Geschlecht," *Neurol. Centralblatt*, 1901.

The text-books teach that tabes occurs much more frequently in men than women: *e.g.* those of Obersteiner and Redlich, v. Leyden and Goldscheider, Gowers, Oppenheim, Grasset. Erb gives 19·5: 1, Fournier 26: 1, Kowshewnikoff 11: 1, Moczutkowsky, quite recently 15: 1.

Mendel's statistics at his Polyclinic in Berlin:—Number of patients 20,539 males, 21,825 females; total 42,464. Of these there were 725 male tabetics, that is 3·53 per cent., and 288 female tabetics, that is 1·31 per cent. There was, therefore, one tabetic woman to 2·7 tabetic men.

He comes to the following conclusion. One can therefore say that the frequency of tabes in the female sex is essentially the same as progressive paralysis among the poorer classes, where about one paralytic woman to every three men occurs; whilst in the well-to-do the relationship is improved five to ten. Tabes occurs at a somewhat later age on an average in women than men, the greatest frequency being from 40 to 45 years. Of the 288 cases, 252 were married women, and thirty-five unmarried. Of the 252 married women, eighty-three were childless, 32·9 per cent. Kron found among thirty-three married tabetics ten sterile, 30 per cent. According to Guttstadt's figures which agree with those of Mendel, 10 per cent. to 15 per cent. of sterile marriages occur among the same classes of non-tabetic married women.

Of seventy female general paralytic cases there were fifty-two married women ; six cohabited with men ; four were juvenile general paralytic cases ; one was imbecile ; seven were single women.

Dr. Bond obtained the very high percentage of 34 out of 70 with certain history or signs pointing to syphilis, and ten doubtful history.

The following data were obtained from statistics kindly furnished me by Dr. Bailey, of Hanwell, relating to 118 female general paralytic cases :—

Of 118 female general paralytics there were 102 married women ; two juvenile general paralytic cases ; three certainly had cohabited ; eleven single, (?) virgins.

The 105 women had 129 children alive, 117 children born alive but dead = 2·3 per cent.

Of these women 34·5 per cent. were sterile, which is almost identical with Mendel's statistics regarding tabetic women.

Dr. Bailey's statistics do not show more than 20 per cent. with a history or signs of syphilis on the body.

This sterility may be explained by the fact that conception had not taken place, or that the embryo had died in the first month or so after conception.

Statistics of Spencer Wells, Simpson and Sims, as well as of Guttstadt, show that married women are childless in 10 to 15 per cent. In female tabetics and paralytics sterility is nearly three times as frequent as the average. In England the average number of children born alive for each married woman is 4·5. It will be found that this corresponds nearly with the male tabetics and tabo-paralytics ; but with the females suffering with this disease it is less than one for each.

That we cannot prove more than between 70 to 80 per cent. of tabic and paralytic patients to have suffered with syphilis is no argument against the doctrine that both paralysis and tabes are post-syphilitic affections. Dr. Crocker has shown that in not more than 80 per cent. of true specific skin diseases can a history of syphilis be obtained. In sixty cases of syphilitic brain disease I could not obtain a history in more than 70 to 80 per cent. The very important experi-

ence of Jadassohn and Hirschl, that only in one-half of the cases of undoubted severe syphilis is it possible to prove primary infection, and the statement of Lang that in one-third of the cases of tertiary syphilis the primary infection was not demonstrable, are arguments against those who will not be convinced that syphilis is the essential cause of tabes and general paralysis unless it can be proved in every case.

Conjugal Tabes and Paralysis.

Mendel, a supporter of the view that syphilis is the most important cause of tabes and paralysis, published in the *Neurologisches Centralblatt* (1888) five cases of paralysis and tabes occurring in married couples; and Dr. Raecke, in the *Monatschrift für Psychiatrie und Neurologie*, vol. 6, published seven of his own cases, and he gives a table with complete literature of sixty-nine cases.

In 38 cases, syphilis certain.
„ 11 „ „ probable.
„ 2 „ „ denied.
„ 18 „ „ unknown facts, but probable syphilis.

Paralysis in both husband and wife occurred in	27 cases.	34 per cent. Tabes in one or other of the couple.
Tabes „ „ „ „	22 „	
Paralysis in man and Tabes in wife	14 „	
„ wife „ „ husband „	6 „	
	69	

This result agrees with Mendel's statement that "if each of the couple suffer with tabes or paralysis, the man is more frequently affected with paralysis." The author also shows that the man as a rule suffers before the woman; the reason is obvious—the man is affected with syphilis first, and is therefore more likely, but not certain to develop the syphilitic degenerative affection. Out of twelve cases of tabes in husband and wife the man was affected first in seven.

Dr. Moenkemoller ("Ueber Conjugale Paralyse bezüglich Tabes," *Monatschrift f. Psych. u. Neurol.*, 1900, Bd. viii.)

found that in 741 paralytics admitted to Herzberge Asylum during six years, there were seventeen cases of conjugal paralysis or tabes. Of these seventeen cases, in fourteen both husband and wife were affected with paralysis; in seven of these, however, one or other suffered with tabo-paralysis; and in two the husband suffered with tabes and the wife with paralysis; in one the husband suffered with paralysis and the wife with tabes. The husband was first affected in thirteen cases; in three cases husband and wife simultaneously; in one the woman was affected first—she was a "puella publica." The proportion showing a history of syphilis is not reliable, owing to the fact that the notes were made at different times by many observers. The statistics are, however, of interest, as showing that the husband is almost invariably affected first; and that the proportion of married couples affected is about 2·5 per cent. This percentage corresponds very closely with the average number of persons affected with para-syphilitic affections.

One of the cases recorded was especially instructive. The husband was infected in 1876. Married in 1879. The wife in 1880 gave birth to a daughter who was syphilitic. In 1894 the husband first showed signs of tabes. In 1895 the daughter first showed signs of paralytic dementia and in 1897 the wife was similarly affected.

My own cases furnish the following results of affection of Married Couples:

Case.		History of Syphilis.	Age and Onset.	Interval between affection of husband and wife.
1	Wife, Ataxy. Husband, Paralytic.	Miscarriages, no living children. Pigmented scar. Indirect from wife's history.	45. Onset with Charcot's joint gastric crises. 35. Death at 36.	10 years, husband affected first.
2	Wife, Tabo-paralysis. Husband, General Paralysis probable, as he had suffered with fits and died insane.	Sterility. Husband, soldier in India.	35. Optic atrophy. Age? Affected after his wife went blind.	?

Case.		History of Syphilis.	Age and Onset.	Interval between affection of husband and wife.
3	E.E.G., Wife, Tabo-Paralysis.	Serpiginous pigmented ulcer on leg. Five stillborn or miscarriages. Four children died in infancy. Seven alive.	62. With mental symptoms. Came on after her husband had been taken to the asylum.	2-3 years, husband affected first.
	Husband, Tabo-Paralysis.	No signs of Syphilis noted in husband, who died at another asylum.	60. Blindness, ataxy and mental symptoms.	
4	Wife, General Paralysis.	Child by first husband, sterile by 2nd (Syphilitic) husband.	35. Worried about husband. Mental symptoms, delusions and hallucinations.	2 years, husband affected first.
	Husband, Tabo-Paralysis.	Scar of chancre and indurated glands, papery scars.	41. Optic atrophy. Ataxy, then mental symptoms.	
5	Wife, Ataxy.	One child born dead.	Ataxy.	Simultaneously or nearly so.
	W.F., Husband, Tabo-Paralysis.	Scar of chancre, indurated glands.	40. Mental symptoms and ataxy.	
6	L.H., Wife, Tabo-Paralysis.	Three miscarriages, said her husband gave her Syphilis.	53. Mental symptoms and ataxy.	1 year after husband.
	Husband, General Paralysis or Pseudo G.P.	.. .. ..	54. Dementia. Spinal affection and paresis.	
7	Wife, General Paralytic.	Syphilitic psoriasis.	37.	Wife admitted to Cane Hill one year after husband had been admitted to Claybury.
	E.C.R., Husband, General Paralytic.	Leuco-placia. Gumma of tongue. Indurated glands.	52. Grandiose delusions, dementia, paresis.	

In all these cases it is probable that the husband infected the wife. In only three cases were the husband and wife both seen. In the remaining four one or the other was seen personally. In all seven it may be presumed that the husband suffered with syphilis and infected the wife; in five out of the seven the symptoms commenced certainly

in the husband first. Syphilis, or signs of it, were observed or indicated in either husband or wife in every case with the possible exception of Case 64 (*vide* p.-m. notes), but the chances of a man being infected who has been a soldier and served in India [1] are very great.

The above cases I have merely come across while making this research on tabo-paralysis, but probably a careful analysis of all the general paralytics admitted into the London County Asylums would show, like Moenkemoller's statistics, conjugal affection in about 2 to 3 per cent. of married general paralytics. This conjugal affection becomes an important argument in favour of (1) the syphilitic origin of the disease; (2) the effect of mental strain and worry in inducing the disease in one of its several forms; (3) the fact that one of the pair may be affected with tabes and the other with paralysis or tabo-paralysis, supporting the view of the unity of the two diseases engendered by the action of the syphilitic poison.

In the *juvenile form of general paralysis* I have found six out of thirty-two in which one or other of the parents had died of the same disease; in at least 80 per cent. of these thirty-two cases, there were signs on the body or a definite history pointing to congenital syphilis, and in the remaining 20 per cent. it was so probable that one might conclude that probably *every* case was of a syphilitic origin. Dr. Tredgold found among his imbecile cases six juvenile paralytics, and all of these were syphilitic cases. Seeing that sexual excesses, alcoholism, and other causes to which general paralysis is attributed must play quite a subordinate part in these cases, it must be assumed that *inherited* syphilis is the essential factor. Three of the total juvenile general paralysis cases (about 10 per cent.) were of the ataxic form, and a considerable number commenced with optic atrophy and blindness long before the mental symptoms developed.

Occasionally one member of a family suffers with tabes

[1] Venereal Diseases in the British Army, *B.M.J.*, July 27, 1901. The Secretary of State for India said that 522 per 1,000 of the British Army had been admitted into hospitals for venereal diseases in 1895, 313 per 1,000 in 1899.

and another with general paralysis, *e.g.* (1) two brothers in two instances; (2) brother and sister in one instance; (3) father, general paralytic; child, congenital syphilis and tabes with optic atrophy. Cases such as these indicate an hereditary soil, or temperament, suitable for the poison to act in producing the degenerative process.

In two of my cases of tabes there was a history of congenital syphilis, and in one at the age of twenty, optic atrophy and absent knee-jerks appeared, which suggested congenital syphilis as a cause, although there was no history of it (*vide* Case 1).

Remak has described three cases of tabes in children; in two there was certain hereditary syphilis, and in one probable; the father of the third likewise had tabes.

Goldflamn has described one case of acquired syphilis which developed tabes and also had several children who suffered from the disease. Strumpell described tabo-paralysis in a 13-year-old girl with hereditary syphilis. Gowers' two cases had both hereditary syphilis. Mendel's two cases both hereditary syphilis. Fournier described four cases, certain syphilis in one, and probable in the other three. Bloc and Gilles de la Tourette each one, with certain syphilis; and Erb three pairs of sisters, six cases of tabes in all, in whom hereditary syphilis was demonstrable. Gowers, Fournier, Raymond, Homen and others have described cases of tabes in adults as the result of hereditary syphilis, and Nonne ("Syphilis und Nerven System," 1901, p. 322) also gives one of his own observations, which is most striking, as follows: "A workman aged 32, who had suffered for two years with lightning pains, and had never been infected with syphilis or addicted to drink, presented all the typical signs of ataxy. He had been treated in the hospital for severe hereditary syphilis." I have occasionally noticed the same in general paralysis commencing in adult life. In my Croonian lectures I remarked that it is possible that some of the cases occurring in adults in which no history of acquired syphilis can be obtained, may still owe the disease to an inherited taint. It is not even necessary that they should show external signs of syphilis, for many of the cases of

juvenile general paralysis were proved to be born of syphilitic parents, although manifesting themselves no external signs of syphilis; whereas their brothers or sisters might exhibit well marked signs. A case of general paralysis died at Banstead Asylum, which had previously been under the care of Dr. Percy Smith at Bethlehem Hospital. The woman had characteristic signs of general paralysis, but she did not manifest symptoms of progressive dementia until she was 30 years of age. The patient was an unmarried woman, and there was no reason to believe that she had acquired the disease.

Post-mortem Table Results in Reference to Syphilitic Residua in Tabes, compared with the Post-mortem Results in Tabo-Paralysis and General Paralysis.

Westenhofer examined the bodies of seventy-two cases of tabes at Virchow's Institute; in 44 per cent. certain or doubtful syphilitic signs were observed. These results compare very closely with those I have obtained in general paralytics; for in 213 male *post mortems* at Claybury Asylum in 1900 and 1901 there were eighty-six general paralytics, and 45·4 of these had signs of syphilis on the body; in the remaining 127, 9 per cent. had certain or doubtful signs of syphilis on the body. In the *post mortems* made on 249 females, there were thirty-six general paralytics; and of them 19·5 per cent. had signs of syphilis on the body. Of the remaining 213, only two (less than 2 per cent.) showed signs of syphilis. The frequency of non-tubercular salpingitis in female general paralytics is also an indication of the greater frequency of venereal infection in these women. It suggests also that a number of these may be recruited from women who had previously led an immoral life. Dr. Bolton's statistics show that out of eighteen general paralytics dying at Claybury fourteen (or 77 to 78 per cent.) showed former non-tubercular tubal disease, whereas in ninety-two other cases only 10 or 12 per cent. showed former non-tubercular tubal disease.

These results are undoubtedly favourable to the syphilitic

view if we consider how often syphilitic lesions heal up in women and disappear, leaving no certain trace behind. In one tabo-paralytic woman there were no external signs on the body; she was sterile. At the autopsy I found a scar on the os uteri the size of a threepenny piece.

Racial Syphilis in Uncivilised Countries in Relation to Tabes and General Paralysis.

It is asserted that in countries where syphilis is extremely common—Bosnia, Herzegovina, Algeria,[1] Abyssinia and Japan[2]—tabes and general paralysis are extremely uncommon or never met with. Egypt was cited formerly, but the statistics of Dr. Warnock, of the Abassieh Asylum, Cairo, show that 6 per cent. of the inmates are general paralytics, and in a recent report he states that syphilis is demonstrable in the great majority of these cases; moreover, he considers it the essential cause of the disease. When I visited Jamaica I ascertained that a large amount of syphilis existed among the negro population; it had become very rife since numbers of them had returned from

[1] At a recent meeting of the Société de Neurologie of Paris, M. Scherb, of Algiers, gave an account of his observations on syphilis and nervous disorders in the Arab, made during three years' residence in Algiers. Tertiary syphilitic symptoms, both cutaneous and osseous, were more frequent among the Arabs than among the Europeans (of Algiers). The rarity of cerebral syphilitic accidents and of para-syphilitic manifestations (tabes and general paralysis) is attributed to the fact that syphilis is a disease of comparatively recent importation among the Arabs of Algiers, and that its stress falls with peculiar vehemence in the tertiary period on the cutaneous and osseous structures. It may be added that the people are essentially lazy, taxing their brain and nervous system but little, and that the organ which is thus least taxed is also the least exposed to the injurious effect of syphilis. The absence of a special predisposition produced by syphilitic or other stresses, however, is not one likely to persist; for with the increase of civilisation and of alcoholism the Arab is preparing himself a neuropathic soil for the future development of both tabes and general paralysis. It is pointed out that other indigenous Semitic races (Jews) present both tabes and general paralysis not infrequently, and they undoubtedly present the neuropathic basis for the development of those affections. In the discussion following the above communication Professor Raymond pointed out that among the Abyssinians syphilis was not uncommon, but it did not produce nervous diseases secondarily. (*Brit. Med. Journal*, Sept. 28, 1901.)

[2] Dr. S. Nose, "Beitrag zur Tabes Syphilis Frage," *Neurol Centralblatt*, 1901. Mittheilungen der Medicin Facultät der Kaiserl. Japanischen Universität in Tokio. Ninety-six cases of tabes dorsalis, eighty-three males and thirteen females. (1) Forty-six cases certainly syphilitic = 47·9 per cent.; ten cases probably syphilitic = 10·4 per cent. (2) Without demonstrable syphilitic infection (*a*) Without gonorrhœa twenty-four cases = 25 per cent.; (*b*) With gonorrhœa fifteen cases = 16·6 per cent.

Panama. Dr. Henderson, of Kingston, informed me that tabes was uncommon; but I found at the asylums many cases of general paralysis; and Dr. Plaxton, the Superintendent, told me the latter was common. I examined there several women suffering with this disease.

On the other hand, in Iceland and the rural districts of Ireland and Sweden syphilis is very rare or unknown, likewise tabes and general paralysis; again, the comparative absence of the diseases, tabes and general paralysis, in priests, Quakers, and women of the better classes in whom syphilis is rarely met with, but where all the secondary or contributory causes occur just as frequently as in other classes of society, tends to support the view that syphilis is an essential factor. We can only reconcile the two contradictory facts that races free from syphilis are free from tabes and paralysis, and that races extensively syphilised do not suffer from these two diseases, by supposing that other contributory factors are absent in the latter, viz., mental stress combined with an emotional, lustful, neurotic, neuropathic or psychopathic temperament, especially when assisted by indulgence in alcohol. Excesses in *baccho et venere* are not only powerful agents in the production of tabes in all its forms, but also in hastening the progress; exposure to cold and wet, also injury, may act as contributory factors. We will now consider each of these causes separately.

Stress.—Prof. Edinger has emphasised the importance of stress in determining the seat of the degenerative process, and he has shown by experiment that degenerative changes in the posterior column of the cord can be induced in animals rendered anæmic by a poison like pyridin, if these animals are daily made to use their limbs for a certain time so as to produce artificially stress of the nervous structures concerned in locomotion. My experience certainly shows some instances in which occupation has apparently determined the seat of the lesion. The occupation of sixty successive male cases of tabes dorsalis met with in hospital practice or outside the asylum, showed only two that were not laborious, involving stress of the

lower limbs. There were two clerks and one of these suffered with cervical tabes, and in the other the arms were early affected and the patient subsequently became a tabo-paralytic case. A mounted policeman was first affected in the arm with which he held the reins. Two packing-case makers, a carpet planner, a parcel-post sorter, were early and markedly affected in the arms. Two tailors were only affected in the legs, but they worked a treadle sewing machine. A carpet planner who knelt all day suffered with tabo-arthropathy of both knee joints, whereas a stone mason who wielded a 4lb hammer in his right hand, and a chisel in his left, suffered with a Charcot's joint of the right shoulder and arthritis of both hands, and of the left elbow. Among the fifteen women a large proportion had to earn their living by charing, which means kneeling or scrubbing, or else they worked a sewing machine ; and this may account for the frequency with which they suffered with tabic-arthropathy affecting one or both knee joints. Of the fifteen women seen in hospital practice, seven suffered with Charcot's joints, and in six out of seven it was the knee. Of the fifty-four men there were only five who suffered with joint affections. Among the tabo-paralytics, there were eighteen in which cord symptoms were known to have preceded mental symptoms, and only two of these did not follow an occupation involving stress on their lower limbs.

Mental worry and strain.—In many of the cases of tabo-paralysis mental worry seems to have preceded the development of the brain symptoms. Not infrequently the blindness from which the patient suffered was a cause. Exposure to cold and wet were often assigned as causes, likewise unequal temperature of the anterior and posterior surfaces of the body, as in men working at a forge, engine drivers and stokers, but as a rule these were only contributory factors of minor importance. According to Erb in only 1·4 per cent. of cases may exposure to cold and wet be considered primary causes. Injury to the spine in four cases was thought by the patients to be the cause of their affection; it certainly seemed that the disease came on after the injury, but would it not have done so without ?

In all the traumatic cases save one there was a definite syphilitic history. Klemperer collected from various sources of literature, thirty cases of so called traumatic tabes, but Hitzig in a valuable monograph has dispossessed Klemperer's inferences of much of their import. Hitzig showed that but few of Klemperer's cases were genuine traumatic tabes. Erb gives the percentage as 0·3, Leyden and Goldscheider say that it is not considerable. It is true that trauma may, as in general paralysis of the insane, act as a contributory factor.

Hereditary Predisposition.—I do not think this plays such an important part in tabes as in general paralysis. Charcot, Ballet, Benedikt, and Borgherini considered it an important factor in tabes; Rosenblatt, Raymond and Fournier, Erb and Gowers as a contributory factor of some importance; Leyden as an effective agent. Redlich attaches no very important rôle to hereditary influence, scarcely more than a contributory factor. In considering hereditary influence we should not only take into consideration nervous and mental diseases in ancestors, but also temperament, which may mean potential neurosis or psychosis. Among the tabo-paralytics and tabic cases suffering with insanity met with in Asylums, 30 per cent. had a family history of insanity. In his Presidential Address to the Medico Psychological Society Dr. Wiglesworth pointed out that hereditary influence plays a much less important part in general paralysis than in other forms of insanity. This was confirmed to me by Drs. Savage, Craig, Spencer, Woods and others.

Intemperance.—In twenty-six asylum cases there was a reliable statement obtained that the patient was not in any way intemperate, and it was concluded that drink as a contributory factor in these cases had no share. In sixteen cases out of sixty it was concluded that drink was a contributory factor of some importance; and in the remainder, no definite reliable statement was obtainable. In a good number of cases the wife's statement was that the patient was a good husband and a good father. I came to the conclusion that drink played a more important part in

tabo-paralysis than in tabes; but that in both it was not such an important factor in the production of the disease as in accelerating its progress. Frequently intemperance and lust is the first evidence met with of the degenerative process affecting the brain or cord.

Mode of Onset of Tabes and Tabo-Paralysis.

Onset of tabes dorsalis. In sixty-five cases the average age at which the onset of symptoms occurred was thirty-seven. The average interval between a definite history of infection and onset of symptoms was ascertainable in more than half of the cases, and was fifteen years. The shortest period was four years and the longest twenty-six. The average age of onset of the disease in the different decades was as subjoined; the age of most cases was determined by the history of some characteristic subjective symptom or group of symptoms, such as pains, double vision, visceral crises, or of the first objective manifestation, &c.:

In sixty per cent., shooting pains were the first symptom which attracted the patient's attention.

In 12·3 per cent., double vision was the first symptom which attracted the patient's attention.

In 14 per cent., visceral, especially bladder troubles, and gastric crises were the first symptoms which attracted the patient's attention.

In 10 per cent., arthropathies or spontaneous fracture.

In 10 per cent., failure of vision and blindness.

The youngest age, excluding a probable congenital case, was 25, the oldest 55.

Decades at which the Onset of Symptoms occurred.

	Tabes Dorsalis.	Female General Paralytics, 118 cases.	Tabo-Paralysis.
1st decade..	1·5[1] per cent.		
2nd ,, ...	1·5[1] ,,	1·75 per cent.	6 per cent.
3rd ,, ...	8·5 ,,	1·75 ,,	4 ,,
4th ,, ...	47·1 ,,	47·45 ,,	72 ,,
5th ,, ...	32·3 ,,	39· ,,	20 ,,
6th ,, ...	13·3 ,,	8·5 ,,	6 ,,

[1] Congenital Syphilis.

Onset of the tabo-paralytic and general paralytic cases was more difficult to ascertain with preciseness, because of the mental condition of the patients, and because of the difficulty to exactly fix the date of onset of mental infirmity; but, generally speaking, the above tables closely correspond, for nearly all the cases begin in the fourth and fifth decades, on an average fifteen years after the period of greatest probability of infection, viz., between 20 and 30. Of fifty-four cases of male tabo-paralysis, the average age of onset of symptoms worked out at $38\frac{1}{2}$. These figures were raised quite one by the existence of a man and his wife whose symptoms (sufficient to attract attention) apparently only commenced at 62 and 63 respectively. The interval of time between infection and onset of symptoms could only be precisely ascertained in fourteen cases; this was partly due to the unreliability of the answers given owing to the mental condition of the patients, and partly, as in the spinal tabetic cases, to the fact that the patients denied, or did not know that they had had syphilis, although there was positive evidence on the body. The shortest interval was six years, the longest twenty-five; average, as in tabes dorsalis, fifteen years.

An argument that is frequently used against syphilis being the cause of tabes or general paralysis in one of these many different clinical types is the fact that there are not more prostitutes affected. Since the great bulk of the cases occur, as we have seen, in the fourth and fifth decades, when it is not so easy for a woman to earn her living by prostitution, she is not described as such upon entering the asylum; and I have found a number of such women in the asylums calling themselves married women, or as following some occupation. Occasionally, indeed, they are described as single women, and in many instances it is subsequently proved (as Case 31) that they have led an immoral life.

Kron's observations upon 184 public prostitutes showed five with tabic symptoms at ages of 27, 28, 47, 47, and 55. But if one reckons only with those over 25 years of age, which allows nine years for the onset of the disease after

infection, there were only thirty-six women, and of these five suffered with tabic symptoms; or 14 per cent. of syphilitic prostitutes suffered with tabes, which is far from a low average. ("Ueber Tabes Dorsalis beim Weiblichen Geschlecht," *Deutsch. Zeitschrift fur Nervenheilkunde*, Bd. xii., 1898.)

In thirty-four cases it was possible to determine the age of onset of the spinal and mental symptoms respectively. Average age of onset of spinal symptoms, the most frequent being lightning pains, was 37½; average age of onset of mental symptoms was 39½. Probably this is too late by several years, for early slight changes of character, irritability of temper, moroseness, &c., are easily overlooked. The longest interval between optic atrophy and mental symptoms was eighteen years. In one half of the cases the spinal and mental symptoms were apparently simultaneous, and the cases frequently, but by no means invariably, ran a rapid course; in some instances they terminated fatally within six to eighteen months of onset of symptoms. In forty-eight cases it was possible to determine whether the spinal signs and symptoms were observed first, simultaneously with, or successive to, the mental symptoms. In twenty-four cases signs of brain affection either preceded or were simultaneous with signs of affection of the cord or eyes, or both. Fournier ("Affections Parasyphilitiques, Tabes et Paralysie Generale," p. 213) says: "Tabes and general paralysis will be different expressions of one and the same morbid entity." He further on asks the question, "Is one authorised to fuse into a single morbid entity tabes and general paralysis?" He sums up the identical relations of the etiology, the close relationship and overlapping in the symptomatology and pathology, and destines them one day or other to be grouped in a single pathological entity.

The foregoing pages on the etiology of spinal and cerebral tabes, clinically distinguished as tabes dorsalis and general paralysis of the insane, tend to show that, pathogenetically, the two diseases are identical. I will next endeavour to show a close clinical and pathological relationship of the different types of a single pathogenetic morbid entity.

Symptomatology.

The fundamental symptoms and signs of tabo-paralysis and of tabes are :—(1) Reflex pupil rigidity ; (2) Lightning pains ; (3) Absence of deep reflexes ; (4) Visceral disturbances, bladder troubles, and gastric crises ; (5) Disturbances of sensibility ; (6) Motor disturbances ; (7) Mental disturbances.

A diagnosis can be made, if the pupil rigidity exists, combined with any of the others, for, practically speaking, reflex rigidity of the pupils to light and pain exists only in tabes, general paralysis, and acquired or congenital syphilis, of which (as pointed out by Sir William Gowers) it may be the sole sign. Very rarely cases of focal cerebral lesions may be accompanied by Argyll-Robertson pupils, especially lesion of the corpora quadrigemina. Some authors point out that in alcoholism and other diseases reflex rigidity of the pupils may exist, but these are cases complicated by syphilis. Indeed, the existence of reflex rigidity of the pupils is evidence that a *mania à potu* is due not only to the toxic effects of the alcohol, but also to general paralysis. The value of this sign is shown by the fact that in thirty-two cases of tumour cerebri found on the *post-mortem* table at Claybury, the notes showed that in only one case was there reflex rigidity of the pupils to light, and this was a syphilitic case with general paralysis. Yet on account of progressive dementia and other mental symptoms, and not infrequently failure to discover or even absence of the cardinal symptoms of tumour (optic neuritis, headache, and vomiting), more than one half of these cases were diagnosed as general paralysis. As a rule, we do not know how long the Argyll-Robertson pupil has been in existence, for, of course, the patient is unaware of its presence, and only comes under observation when some other early symptom such as pain, a fit or mental symptoms, double or defective vision, visceral crises, or ataxy brings him to the doctor.

Eye Symptoms of Tabes and Tabo-Paralysis.

Pupil changes are met with in nearly all cases of tabes and tabo-paralysis, and in the majority of cases of general

paralysis, in the earlier or later stages of the disease. The hospital cases of tabes closely correspond to the asylum cases of tabes and tabo-paralysis as regards pupil phenomena, as the subjoined statistics show.

Tabes (Hospital Cases).			Tabes or Tabo-Paralysis (Asylum Cases).
73·5 per cent.	Argyll-Robertson pupils on both sides	..	70 per cent.
3 ,,	,, ,, one side	..	7 ,,
3·7 ,,	sluggish to light	..	4 ,,
15 ,,	inactive to light and accommodation[1]	..	20 ,,

Occasionally in tabes and more often in tabo-paralysis and general paralysis, paradoxical reaction to light occurs; viz.: on removal of the shadowing hand no reaction takes place for a second or two, then the pupils dilate slightly. Möbius explains this phenomenon thus: that probably during the process of covering the eyes, the patient is accommodating, and when the shadowing hand is removed, accommodation is relaxed, the process of accommodation being a slower process than the light reflex. A sluggish reaction to light is not necessarily pathological, and unless there is a difference between it and accommodation, it may be of no import. With absence of the light rigidity, there is nearly always absence of reflex cutaneous pain, and it is not uncommon to see a patient suffering agonising pain with pin point pupils.

Inequality, Irregularity and Size of Pupils.

Before the discovery of the Argyll-Robertson phenomenon, inequality of the pupils afforded a valuable sign of the disease; likewise very small pupils; and even now such alteration may attract the physician's attention, and lead to his making a more careful examination of a case which presents urgent symptoms pointing to organic disease of internal organs; *e.g.*, in Case 7, which was admitted for acute intestinal obstruction, the surgeon who was called in to operate had his attention attracted to the very small

[1] Most of the cases which were totally inactive to light and accommodation were the subjects of complete optic atrophy.

pupils, while the man was suffering agony, and this led to the discovery of the true nature of the disease. Inequality of the pupils is more frequently met with in tabo-paralysis and general paralysis than in tabes.

Size, Inequality, Irregularity of Pupils in Tabes (Hospital).		Size, Inequality, Irregularity of Pupils in Tabo-Paralysis (Asylum).
Unequal [1]	40 per cent.	... 65 per cent.
Irregular	18·5 ,,	... 27 ,,
Small	32 ,,	... 33 ,,
Pin Point	18 ,,	... 16 ,,
Medium	34 ,,	... 33 ,,
Large on one or both sides	16 ,,	... 18 ,,

Paralysis of the external ocular muscles may be transient or permanent. Diplopia (double vision) is often the first subjective symptom which attracts attention; but it may be due to a syphilitic meningitis. In several of my cases it occurred within a few years of the syphilitic infection, but as a rule the interval is much longer. Any muscle may be affected, but the external rectus is the most frequent. The patients often complained of double vision, which lasted a few days or weeks, for which they attended an eye hospital long before the other symptoms troubled them. Some of these cases were sent on to me from the Westminster Ophthalmic Hospital, or by my friend, Mr. Treacher Collins, from Moorfields.

Ocular paralysis in the hospital cases, a considerable number of which, however, were sent from the eye hospitals, render the following statistics much too high.

In twenty-two there was double vision of a transitory nature; it was, in nearly all the cases, a very early symptom. There were nine cases of ptosis (in four associated with squint) and four of

[1] These figures are only approximate, as a number of cases were only seen once or a few times. The series contained a considerable number suffering with optic atrophy or blindness. Irregularity of the pupils may occur independently of synechiæ, the pupil being angular, elliptical or oval; it was more frequently met with in the asylum than hospital cases. The asylum cases, as a whole, came more frequently under my observation, and I was able to note the fact that the degree of inequality varied considerably and, my impression is, much more frequently than in the hospital cases of Tabes. Not infrequently a small pupil became large or medium-sized, and this often occurred during or after a series of fits. Frequently pupils which on the one hand were equal at one period of the disease, became unequal, while on the other hand, pupils which were unequal might become nearly equal.

nystagmus. In eleven cases there was permanent ocular paralysis, four partial of 3rd, one of 4th, five of 6th, one ophthalmoplegia interna and externa bilateralis.

Among the asylum cases, paralysis of the ocular muscles was much less frequently met with. In only eight was double vision described as an early symptom (13·3 per cent). In three cases transitory diplopia occurred while the patient was in the asylum. Permanent ocular paralysis occurred in four cases, two unilateral ptosis, and two unilateral 6th nerve paralysis.

Atrophy of the optic nerve is one of the most serious symptoms that can arise in tabes; it is an early, indeed a very early symptom, and the defect of vision or blindness may be the first cause of the patient seeking advice; its frequency is difficult to ascertain. Sir William Gowers states that one in ten cases suffers with it; according to Leimbach it may be the first symptom in 1·5 per cent. Among my sixty-five hospital and infirmary cases, there were twenty with optic atrophy and ten of them were completely blind. A great many of them remained in the pre-ataxic stage many years (*vide* Cases 1, 61); others became general paralytics and died. The large number of these cases was due to the fact that a considerable number were sent to me from Ophthalmic Hospitals. Again, if we took infirmary cases it would hardly be fair, because blindness leads to incapacity to earn a livelihood. When, however, we come to consider asylum cases we find that it is extremely common in tabo-paralysis; owing, however to the difficulty in examining these patients ophthalmoscopically a few cases may be overlooked, but 35 per cent. of the sixty cases were found to have well marked optic atrophy, and 50 per cent. would probably be nearer the mark, for I have found often, *post mortem*, optic atrophy which was not noted during life. In 150 consecutive cases of general paralysis found on the *post-mortem* table at Claybury, the optic nerves were carefully examined, and naked eye atrophy was found in only 7 per cent.; several of these belonged to the juvenile form of general paralysis (in which it may occur independently of any degeneration in the posterior columns of the spinal cord), the remainder belonged to the tabetic form of the

disease in adults, as ascertained by microscopical examination of the spinal cord. Optic atrophy, then, occurring in tabes is to my mind, apart from the blindness, a serious indication of the possibility of the degenerative process attacking the brain. My experience would lead me to believe that the mental worry occasioned by the blindness in a few instances tended to bring this about. The failure of vision usually commences with limitation of the peripheral field of vision in one eye and loss of colour vision; then the other is affected, or both may be affected simultaneously. The onset is usually gradual and the course slowly progressive, but sometimes the sight is lost, apparently in a few days, or almost suddenly. Doubtless in some of these cases there has been loss of the peripheral field of vision with the retention of central acuity, and it is the comparatively sudden destruction of the remaining fibres to the macula which leads the patient to believe he has suddenly lost his sight. It may, however, be (as Gowers suggests) due in such cases to an interstitial inflammatory process, but ophthalmoscopic examination shows no change to account for it.

Dr. Wiglesworth was one of the first in this country to call attention to optic nerve atrophy *preceding* the mental symptoms of general paralysis of the insane. In conjunction with Mr. Bickerton, he described the optic nerve changes met with in a series of sixty-six cases of general paralysis. They showed that while in the majority of cases of this disease the fundus oculi presented a normal appearance, in a considerable minority changes in the direction of neuritis or atrophy were found.

The credit of first describing the form of tabes known as optic tabes is due to Benedikt, who stated in 1881 that the abortive cases of tabes (*formes frustes*) are the ones in which optic atrophy is a prodromal symptom.

In 1887 Benedikt stated a law, from which he knew no exception, that the tabetic motor symptoms, no matter what development they may have reached, vanish as soon as optic atrophy appears. Dejerine points out that this latter statement is not true and I certainly agree with him, *vide* (among many others Case 28.) I would go further and say that

it has no influence upon the ataxy, when it arises in a patient already in the second stage of ataxy; in this respect it agrees with the fact that cortical degeneration occurring in an advanced case of tabes with *well marked* ataxy does not cause the disappearance of the ataxy, although the advent of dementia and other signs of cortical degeneration like optic tabes certainly arrests the spinal degenerative process and modifies ataxy of the first degree. An explanation of these facts is attempted in the chapter on "Inco-ordination." Dejerine states that the number of cases of tabes with blindness in which the disease does not progress beyond the pre-ataxic stage is considerable. He and Martin studied 100 tabetic cases at Bicêtre, eighteen of whom were completely blind, and none of these presented motor disturbance. According to my experience lightning pains usually precede optic atrophy, but not invariably. Usually when the optic atrophy occurs early, the lightning pains diminish in intensity, and even at times disappear.

An important point to remember is the frequency with which optic atrophy is followed by tabo-paralysis; in quite 50 per cent. of the cases of this form of the disease met with in asylums (and they are fairly numerous), the cerebral symptoms followed optic atrophy and blindness. In many instances the mental trouble occasioned by the blindness and loss of livelihood undoubtedly acted as the exciting cause of the brain degeneration.

Patients with paralytic dementia on the one hand, do not as a rule suffer with visual hallucinations, except in the acute maniacal condition, especially that accompanied by alcoholism. Cases of tabo-paralysis, on the other hand, with optic atrophy frequently have visual hallucinations (*vide* p. 79.)

Sir William Gowers states that Charcot believed that nearly all cases of so-called simple primary optic atrophy *develop finally* spinal symptoms, and he reports a case in which amaurosis lasted twenty years before any other symptom of tabes was noted; in another case sixteen years passed before distinct signs of tabes occurred. Every case of optic tabo-paralysis that I have examined *post mortem*, although many of them showed no ataxy during life, yet

presented upon microscopical examination well marked degeneration of the posterior columns due to degeneration of the exogenous system of fibres, and corresponding atrophy of the posterior roots (*vide* Cases 61-63).

Section II.

Sensory Disturbances.

Sensory disturbances are either subjective or objective, and these again are of two types: (1) destructive, causing loss of function; (2) irritative, causing abnormal or perverted function. The sensory disturbances relate to common sensibility of the skin, viz.: thermal condition, pain and pressure, or to the special senses, especially the complex of sensations termed "kinæsthesis" or muscular sense of Bell. The clinical phenomena of tabes, and, in a measure, of tabo-paralysis, are due to these disturbances. Although there is a symptomatic similarity among all the cases by the more or less constant presence in some stage of the disease of certain sensory disturbances, *e.g.*, lightning pains, anæsthesia or analgesia, which are the best examples of the two types, there is hardly ever identity of symptoms, and no two cases are exactly alike. The complexity of the symptomatology agrees with the polymorphic character of the lesions. A study of the microscopic changes in the peripheral and central nervous systems in a large number of these cases explains this general uniformity and special diversity of the symptoms. The fact that changes always occur in the posterior spinal roots and their projections in the cord explains the general uniformity in the objective and subjective sensory disturbances of the skin, muscles, and joint structures, whilst the variability of distribution and extent of the morbid process in thirty-two posterior roots, and their projections in the cord, as well as the variable degree of complication by lesions of the peripheral afferent nerves, explains the frequent diversity of precise distribution of the anæsthesia, analgesia, hyperæsthesia, hyperalgesia

met with. The sensory dissociation shows that certain fibres subserving special functions are particularly affected, *e.g.*, the muscular sense-fibres of the lower limbs, and the nerve-fibres innervating joint structures; again the light tactile anæsthesia of the mid-thoracic region, without analgesia, shows that the disease process is selective, and is related to the intraspinal terminations of the roots and not the peripheral nerves. For, in a neuritis, the sensory symptoms have not this dissociation, nor this distribution. The existence of these sensory disturbances in the insane tabetic, helps to explain many of their delusions and illusions, *e.g.*, the idea that enemies are torturing them with electricity (it was hot irons and pincers before electricity was in general use).

The subjective disturbances of the nerves of special sense and of the viscera are still more liable to receive an insane interpretation, and give rise to delusions and illusions. There is a definite relationship between the degree of ataxy and the reflex spinal tonus, which is due to partial or complete abolition of the intraspinal paths conducting afferent impressions from the muscles, tendons, and structures around and within the joints, and this is due not only to the withdrawal of the guiding sensations, but also to muscular hypotonus.

The skin sensibility was tested in forty-eight successive cases of tabes and a number of cases of tabo-paralysis, and mapped out on charts; the distribution was subsequently, when the series was completed, compared with the root distribution given by Seiffer, which is based upon an analysis of the researches of Head, Thorburn, Kocher, Starr, and Wichmann. From thirty-two cases where root distribution was sufficiently definitely determined as regards tactile anæsthesia, a composite chart was made.

Method of Examination.

Anæsthesia to light tactile sensations was made by touching the patient very lightly with the tip of the finger, analgesia by pricking with a needle, and heat or cold by a

test-tube filled with ice or hot water. The patient's eyes were covered with a towel. Twelve of these cases were carefully noted by my clinical clerk, Mr. J. P. Candler, who embodied the results in a thesis. Most of these, however, I myself have verified. The distribution and character of the sensory disturbances, when combined with a careful examination of the spinal cord and roots, will help to throw some light upon the conduction of various sensations in the cord, the functions of various tracts and groups of fibres, also the inflow of visceral sensations by the white rami. The spinal cords which I have systematically examined so far have been mostly obtained from the cases of tabo-paralysis or cases of tabes in which the sensory disturbances were not accurately determined during life, owing either to mental disturbances, or to the fact that they died before I commenced these researches of the accurate distribution of the sensory disturbances. Before passing to a description of my own observations, I will give a brief description of the results of other observers.

Duchenne, "Traité d'électrilization localizée," 1872, devotes a few lines to the sensory manifestations of tabes. He mentions that in the second period of the disease abolition of tactile and painful sensibility occurs. The sensibility of the feet and hands is in general more or less diminished, especially the palmar and plantar surfaces following the appearance of troubles of coordination of movement. In the skin the painful and tactile sensibility are simultaneously damaged, but more often painful sensibility is intact or little altered, and temperature is last affected. Duchenne, therefore, noted the sensory dissociation, although he was incorrect in several other ways.

Topinard, "De l'ataxie locomotrice," 1864, also Cruvielhier had noticed this sensory dissociation.

A most valuable paper has recently appeared by Förster and Fränkel.[1] These authors give an admirable summary of the work of previous observers before describing their own observations. They point out that Oulmont in Charcot's Clinique was the first to show that the statement of Duchenne regarding the fact that the objective disturbances were most constant in the extremities,

[1] "Untersuchungen über die Störungen der Sensibilität bei der Tabes dorsalis" von Dr. Fränkel und Dr. Foerster. *Archiv f. Psychiatrie und Nervenkrank.* Band 33, Heft 1.

was incorrect. Oulmont examined twenty tabetic women and found sixteen cases of trunk disturbances of sensibility. These disturbances were especially localised between the nipple and umbilicus and characterised by their symmetry.

The essential subjective phenomenon is girdle-sensation, and below the girdle of anæsthesia (at least in front) is a zone of hyperæsthesia.

Hitzig first emphasised the great frequency, and above all the diagnostic importance, of these sensory disturbances of the trunk.

Lähr, whose researches include sixty tabetic cases, found sensory disturbances of the trunk fifty-five times. The five cases where these sensory disturbances were absent were five tabo-paralytics, with only slight but nevertheless distinct spinal symptoms.

Summary of Lähr's results :

(1) Hyperæsthesia appears to be a regular and generally early sensory disturbance of tabes.

(2) The sensory disturbances for a long time consist only of a diminished sensibility for light tactile impressions, whilst, as a rule, in the legs there is at the commencement only a diminution of painful sensations and of the sense of position. The latter generally appears to precede the trunk hyperæsthesia.

(3) The localisation in the trunk usually corresponds to the distribution of the middle or lower dorsal nerves. Their further distribution follows generally pretty symmetrically the encircling horizontal zone of the trunk, which extends upwards and downwards and in a characteristic manner, to the arms. In sixteen cases it spread to the arms, first to the axilla, then to the ulnar side of the arm and lastly to the radial.

(4) The extent of this tactile anæsthesia is quite characteristic, it corresponds not to the area of distribution of the peripheral, but to that of spinal roots and their intra-medullary projection fibres. It is not, however, contended that the peripheral nerves may not be affected.

(5) A wide-spread hyperalgesia, especially for cold, occurs in the borders of the hyperæsthetic or anæsthetic areas and between the zones affected; and the reflex excitability of the skin is here very lively. In the hyperæsthetic and anæsthetic area it may be lowered or lost.

(6) Sensory irritation phenomena are very common, but not a regular accompaniment of the anæsthesia. Ulnar compression causing no tenderness appears as a rule in tabes in conjunction with other demonstrable sensory disturbances.

This admirable piece of work by Lähr was an important addition to our knowledge of the sensory disturbances of the trunk and especially to the localisation of the sensory disturbances in the upper extremity. Lähr also pointed out the existence of perfectly normal areas of sensibility lying between hyperæsthetic or anæsthetic areas.

Chipault's researches on the topography of sensory disturbances in fourteen cases of tabes showed similar results to those of Lähr.

These areas according to Chipault are especially the seat of sensory anomalies, the trunk, the arms, the legs. In more than one-third of his cases there was a continuous sensory disturbance of arms, trunk and legs, which is in opposition to the results of Lähr, Förster and Fränkel.

Probably his series included a large proportion of cases in the third paralytic stage. He lays stress on the coincidence of the subjective phenomena, pains and paræsthesia, with the objective demonstrable anæsthesia and analgesia of the several areas.

Patrick made observations especially upon the trunk-anæsthesia, and he called attention to the fact that in the trunk there occurs almost always only pure tactile anæsthesia, which is consequently regarded by him as a special sensory disturbance. He also pointed out that unaffected areas lie between affected ones.

Hintze described trunk anæsthesia in six out of seven cases.

Marinesco examined fifty cases in the cliniques of Raymond, Marie, and his own at Bucharest with the following results. There are four principal regions in which there are light tactile disturbances of sensibility.

(1) Thoracic.—Forty out of fifty cases.

(2) Genito-perineal.—Under side of scrotum and later the whole scrotal regions, in well marked cases, also the penis. Less frequent in the region of the anus.

(3) Region of the upper limb, ulnar side.

(4) Region of the lower limb.

It is difficult to estimate a precise type for the lower limbs, for here are the greatest variations. Four regions correspond to well-pronounced sensory subjective disturbances: anæsthesia of the thorax to the girdle sensations: genito-perineal to impotence and rectal crises; anæsthesia on the inner surface of the arm to the ulnar sensation; lastly, anæsthesia of the lower extremities to the lightning pains, and paræsthesia.

Förster and Fränkel. These authors give a brief description with charts of fifty cases especially relating to sensory disturbances of the skin, joints and muscles. Their conclusions are as follows:

(1) No single case of tabes with ataxy in which the sense of position of joint was not affected. The authors speak of it as joint sensibility.

(2) Generally speaking, there is a parallelism between the sensory disturbance and the ataxy, but individual differences may exist according as the cerebrum reacts to the imperfect sensory stimuli. The degree of ataxy may not be the same in the two limbs, and the joint sensibility will then be more impaired on the side most ataxic.

(3) As a rule, the disturbances are most marked in the joints of the toes and ankles; but sometimes they may be more marked in the joints of the hips and knee.

(4) They call attention to the great frequency of the joint sensory disturbances in the upper limb, generally not marked, and much less (usually) than in the lower. When the case is one of arm tabes, shoulder, elbow, and wrist are affected; otherwise only the fingers (twenty-three times out of thirty-two tested cases).

(5) They determined the loss of sense of fatigue in five cases.

(6) *Skin Sensibility.* In not one of the forty-nine cases studied could the skin sensibility be considered normal. In all but five of thirty-eight cases in which there was light tactile sensory disturbance, there was also present disturbance of painful sensibility. In the remainder there was a disturbance of painful sensibility.

Topography of disturbances of skin sensibility :—

Sensory disturbances were found in the face in six cases arising at all stages of the disease. In one case, the one half of the tongue and the mucous membrane of the mouth on one side.

Sensory disturbances of the trunk occurred in forty-five cases out of forty-nine; in one case the patient was in the pre-ataxic stage. The anæsthesia generally begins above the axilla and extends forwards through the nipple to the middle line. The upper and lower borders are horizontal, uniform lines, but considerable deviations from this, the usual form, may exist, there being occasionally only islands of anæsthesia or hyperæsthesia. Out of thirty-eight cases in which light tactile disturbances were discovered, in only eight was analgesia or blunting of painful sensation found. Loss of painful sensation of trunk is later than light tactile sensibility, and in only a single case did they find the area of painful sensation more extensive than that of tactile. In four cases the anæsthetic area was hyperanalgesic. Very frequently there was an increase in the sensory excitability in the

neighbourhood of the anæsthesia. Hyperæsthesia of the trunk for cold was frequently present, also sensory disturbances of the upper extremity.

In thirty-seven cases out of forty-nine the arms were affected. The anæsthesia was continuous with the trunk anæsthesia, and extended as a streak along the inner side of the arm, limited sometimes to the upper arm and often extended to the fore-arm, and not infrequently to the inner side of the ring finger and the whole of the little finger. It may extend to the whole fingers, but very rarely affects the whole arm. The sensory disturbance is usually anæsthesia or hyperæsthesia.

Sensory disturbances of the lower extremity :—

In forty-four out of forty-nine cases there were disturbances of sensibility in the lower extremity ; in eighteen of these, disturbances round the anus. In all cases the sensory disturbance was analgesia or hypalgesia, and if tactile disturbance was present, it was usually less extensive than the analgesic area, thus contrasting with the sensory disturbance met with in the trunk and upper extremities. Disturbances of heat and cold sensations are rare.

Having thus given a brief account of previous observations, I will now proceed to give the results obtained in my own cases.

Subjective Sensory Phenomena of Tabes and Tabo-Paralysis.

Lancinating Pains.—The pains of tabes are variable in intensity, situation and duration. The patient usually has suffered months or years before their true nature is discovered. They may be slight or severe, generally occurring in paroxysms, and are likened to stabbing, shooting, boring, or lightning, or to hot wires thrust into the flesh. A patient may be free from pains for hours, days, weeks, or even longer ; they may last a few minutes and then cease, to recur again in the same situation or in another. The attacks of pain may last a day, a day and night, or several days, causing the patient the greatest suffering and agony, relief being obtained only by morphia. Rarely does the pain correspond with the distribution of the nerve, although a patient may come thinking he is suffering with sciatica. The seat of the pain is most varied. It may start in the

great toe, and the patient thinks he has gout; it may start in joints and be attributed to rheumatism; or it may be felt in the skin or muscles. The skin is usually hyperæsthetic over the seat of the pain, whether it be superficial or deep, and this hyperæsthesia may last after the pains have ceased. Herpetic eruptions follow in very rare instances, for I have only met with one case in sixty, and that subsequently turned out to be a tabo-paralytic. In one case of tabo-paralysis symmetrical bullæ occurred in the limb and trunk. Examination of the peripheral nerves showed acute degeneration of the fibres, proceeding to the skin area of the bullæ. Also acute degeneration changes in the cells of the corresponding spinal ganglia. In another patient erythematous patches occurred after an attack of pain in the arms. Some patients complain of pain running in the long direction of the limbs, which they mistake for sciatica. When pain occurs in the trunk it is frequently unilateral, and not uncommonly accompanied by gastric crises. The girdle pain is frequently experienced, and a tightness compared to an iron jacket or the constriction of a tight belt has been complained of. The pain running down the inner side of the left arm might be mistaken for angina. Very often the pains radiate all over the body, and quite a number of patients who suffered with gastric crises said that with the attacks of vomiting, pains started from the mid-thoracic region and radiated all over the body with the exception of the face. One man pointed out that the face was unaffected, except in an area which corresponded to the distribution of the second cervical root (*vide* fig. 1, p. 47); another, a woman (Case 17), suffered so severely that she could hardly bear the touch of the bed-clothes, and even the light of the windows was so painful she would bury her face in the pillow; the only part of the body where she did not suffer the severe pains during these crises was the left leg below the knee. The right leg had been amputated above the knee for Charcot's joint. The left leg, in which the pains did not radiate, was absolutely analgesic and anæsthetic below the knee. This would rather indicate that the sentient gray matter subserving painful sensation for this leg or the intra-

spinal terminals of the posterior spinal neurones had been completely destroyed, therefore painful sensation could not be projected outwards into the limb. That this is a more likely explanation than destruction of the peripheral nerves is shown by the fact that pain of central origin may be referred to a limb which has been amputated.

Another case was interesting as showing that a toxic condition of the blood may set up crises and attacks. This patient (Case 20) had a discharging sinus from a suppurating Charcot's joint; she volunteered the statement that when pus accumulated owing to the sinus getting blocked, she was pretty sure to have an attack of vomiting associated with severe pain.

In the tabo-paralytics or tabetics with mental symptoms these attacks of pain may be insanely interpreted. They are, therefore, rather illusions than delusions. These patients often believe they are being tortured by unseen agencies, that electricity has been turned on by their enemies; they have been given poison which has gone into their legs and feet (as Case 66). They may associate the pains experienced with dreams or visual hallucinations; and they may tell you (as in two cases) that lions and wolves came and gnawed their limbs by night, and will beg you not to let them be tortured again. The pains may last during the whole course of the disease, but frequently in the third stage of the disease, when the roots which are undergoing degeneration have become completely destroyed, the pains abate or cease; that is if the disease does not steadily spread up to affect the arms. In tabo-paralysis dementia is usually associated with abolition of the painful sensations caused by pricking, and the patient as a rule does not complain of attacks of pain or torture; in this respect, markedly differing from the tabetic with delusional insanity, who probably suffers more than the sane tabetic, as he is not only tortured with physical pain, but also with delusions of persecution by unseen agencies—the true pains forming a realistic basis to the delusions around which his whole psychical existence may centre. Nearly all my cases of tabes with mental symptoms (twelve in number) were the subjects of

inherited insanity, and we may consider these cases as tabes occurring in persons potentially insane—the organic disease, with its complex symptoms, being sufficient to act as a determining factor of insanity.

Paræsthesia.

Subjective sensations of various kinds, as numbness, pins and needles, formication, a cold trickling feeling in the skin, a feeling in the soles of the feet of walking on putty, wool, or velvet may be complained of. In rare cases Hutchinson's mask, due to affection of the fifth, occurs. The patient says his face feels stiff, and he feels as if it were covered with a cobweb. Paræsthesia of the arms, like the pains, affects the post axial border of the limb, the distribution of the (eighth) cervical and first and second dorsal roots. Consequently patients often complain of numbness and tingling in the ring or little fingers (*vide* figs. 1 and 2, p. 47). The insane tabetic may put a false interpretation upon these abnormal subjective sensations, *e.g.*: Case 31 said mice were running over her in bed at night, and she saw them on the pillow. Another used to get up at night and brush the flies out of his bed. Another (Case 27) had a feeling of cold water running up his back and over his head as far as the forehead, but not on to his face. The subjective sensation he insanely associated with the medicine he had had given to him from the London Hospital, which he asserted took the course in his system of this subjective sensation. It was of interest to learn from this patient that he had been warned by another patient that if he repeated this statement before the magistrate he would be sent away to the asylum. "But he had to speak the truth"—so convinced was he that the medicine was the cause of his trouble.

Objective Sensory Disturbances.

Forty-eight successive cases of tabes were carefully tested for cutaneous sensory disturbances. In forty-two cases, objective cutaneous sensory disturbances were found; the six cases in which no cutaneous disturbances were discovered

were all in the pre-ataxic stage. Several of these pre-ataxic patients had had, however, subjective sensory disturbances. There were two other cases in the pre-ataxic stage in which gastric crises were associated with light tactile sensory disturbances of the mid-thoracic region and no sensory disturbance elsewhere. Trunk anæsthesia to light tactile impressions is the earliest and most constant sensory disturbance. In thirty-six cases there was anæsthesia of the trunk to light tactile sensation, and of these thirty-six twelve suffered with gastric crises. No patient suffered with gastric crises that did not develop sensory trunk anæsthesia. In most cases the affection was bilateral and symmetrical or nearly symmetrical, both upper and lower borders forming a sharp horizontal line which corresponded to the zones of distribution of the roots shown in the accompanying diagrams, Figs. 1 and 2. In this zone of anæsthesia to light tactile impressions, there may be a zone of analgesia or hypalgesia, or scattered points of blunted painful sensation. The area of anæsthesia or hypæsthesia of the trunk was usually more considerable than the analgesia or hypalgesia. The zone of hypæsthesia or anæsthesia is occasionally only unilateral, or it may be asymmetrical extending over more segments on one side than the other. There may be one or several segments anæsthetic, the most frequent being the fourth and fifth. Sometimes a segment above or below is hypæsthetic. In two cases the area supplied by a dorsal branch or branches of the posterior root was unaffected, while the area supplied by the anterior was anæsthetic. In one instance (Case 16) the patient was anæsthetic only over an area supplied by the posterior divisions of the fourth and fifth roots of the right side; then after an attack of severe pains in this region accompanied by bilious vomiting, the area supplied by the anterior divisions became affected and eventually a complete girdle of anæsthesia was found to exist. The trunk anæsthesia is most frequently met with in the fourth and fifth segments; it may extend up the chest to the third or the second interspace, and then the inner side of the arm also becomes affected. This may extend to the whole inner side of the arm, or only the inner side of the upper arm and fore-

arm. In advanced cases, or cases commencing in the arms, there may be light tactile anæsthesia continuous with the thoracic anæsthesia affecting the whole inner half of the arm, and corresponding to the distribution of the second dorsal, first dorsal, eighth cervical and seventh cervical—that is, the whole post-axial border of the limb. It does not extend beyond the second intercostal space on the trunk,

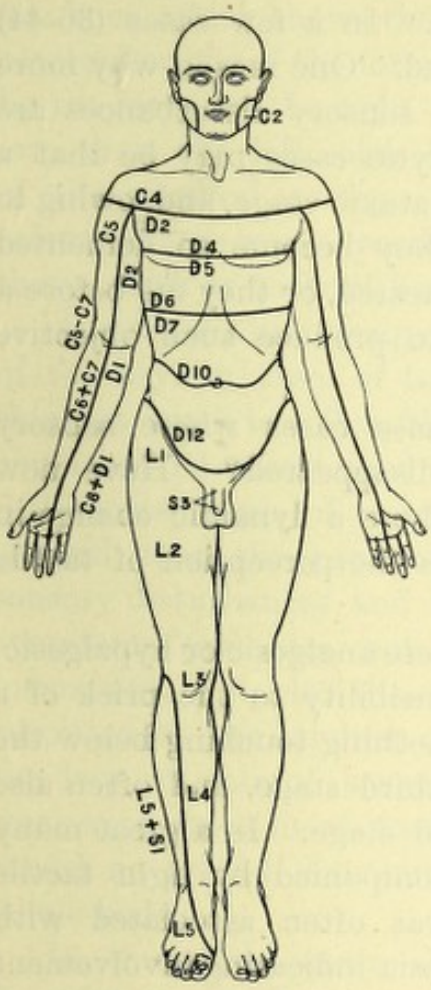


FIG. 1.

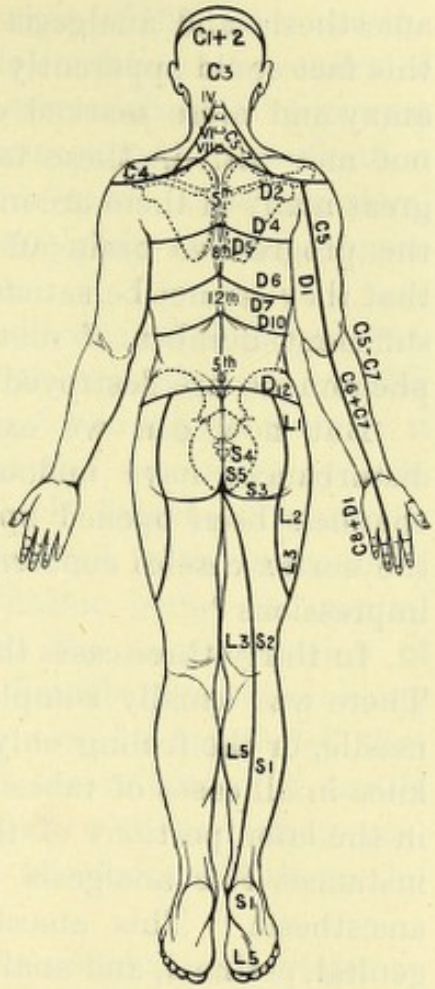


FIG. 2.

Distribution of posterior roots to the skin, after Seiffer.

because it is very seldom that the upper cervical roots which enter into the formation of the cervical plexus are affected. In one very advanced case of tabes the whole of the skin distribution of the brachial plexus was anæsthetic. In this case (17) there was continuous anæsthesia from the fifth cervical to the fifth sacral, inclusive. In four cases the

cutaneous anæsthesia of arms, trunk and legs was continuous. In twelve cases the arms were affected, but only three times did the anæsthesia extend to the radial side of the median line. Whilst a considerable number of the tabo-paralytics showed subjective sensory phenomena at some period of the disease, in only some cases (31, 45, 46) could objective phenomena be demonstrated; indeed the appearance of the mental symptoms not only diminished the ataxy, but the anæsthesia and analgesia as well. In a few cases (36-44) this fact could apparently be proved. One reason why more ataxy and more marked objective sensory disturbances are not met with in these tabo-paralytic cases may be that a great many of them are in the pre-ataxic stage, and, owing to the progressive brain affection, they become so demented that they cannot be satisfactorily tested, or they die before a sufficient number of root-fibres to produce such objective phenomena are destroyed.

But how can we explain those cases where sensory disturbances have undoubtedly disappeared? Have new channels been opened up, or is there a dynamic change in the cortex cerebri concerned with the perception of tactile impressions?

In thirty-three cases the legs were analgesic or hypalgesic. There was usually complete insensibility to the prick of a needle, or the feeling only of something touching below the knee in all cases of tabes of the third stage, and often also in the later portions of the second stage. In a great many instances this analgesia was accompanied by light tactile anæsthesia. This anæsthesia was often associated with genital, perineal, and anal anæsthesia indicating involvement of the lower four sacral roots, whereas the parts below the knee indicate lumbar four and five and sacral one. Analgesia or hypalgesia of the lower extremities is sometimes limited to the sole of the foot or the peroneal surface of the lower part of the leg; it may exist independently of trunk anæsthesia and be the sole objective evidence of sensory disturbance. A patient may previously show no cutaneous disturbance and then anæsthesia or analgesia develops after several attacks of pain.

Analgesia in tabo-paralytics is fairly common, no response may be elicited from the sharp pricking of a pin all over the body (Case 55); or the patient may exhibit a cutaneous sensory disturbance of two-fold origin, *e.g.* Case 45, where there existed a hemi-anæsthetic condition of the limbs on the left side in which there was hemianopsy, and successive epileptiform seizures, leaving the other right half of the body free, with the exception of some analgesia below the knee, which shows the spinal origin of the latter and the cerebral origin of the former. This condition was confirmed by the fact that the right hemisphere weighed 100 grammes less than the left, also by the microscopical examination of the cerebral cortex. In other cases after unilateral seizures there was a partial hemi-analgesia and hemi-anæsthesia, with or without hemiparesis.

Anæsthesia and analgesia were discovered in 42 per cent. of the asylum cases of tabes and tabo-paralysis; probably this was much less than the reality, for in a good number it could not be ascertained owing to the mental symptoms. Still some cases of ataxy, in which definite information showed that the patients had suffered with pronounced sensory disturbances and inco-ordination before the mental symptoms came on, not only lost the inco-ordination of movement, but also the sensory disturbances, after being admitted to the asylum. It may also be mentioned that the loss of painful sensation and the retention of tactile was, as a rule, more frequently met with, and often possessed no special distribution, but affected one half or the whole body.

In thirty-two cases the charts illustrating cutaneous disturbances in the form of anæsthesia corresponded fairly accurately with the distribution of definite posterior root areas in accordance with figs. 1 and 2, and the accompanying diagrams (figs. 3 and 4) were constructed from them.

Hyperæsthesia and hyperalgesia.—An area in which pains have been experienced, whether on the trunk or limbs, may be hyperæsthetic, and precede anæsthesia or analgesia; it indicates the irritation prior to destruction of the root-fibres or their intraspinal terminals. Very frequently an

area may be hyperæsthetic above or below a complete anæsthetic or analgesic area. A zone of hyperæsthesia is therefore frequently met with above the third or fourth thoracic interspace, or below the seventh or eighth. Again, there may be a patch of hyperæsthesia situated within an anæsthetic area; or on one side of the trunk, or in one limb, there may be hyperæsthesia or hyperalgesia, while corresponding parts, or nearly corresponding parts, of skin on the other side

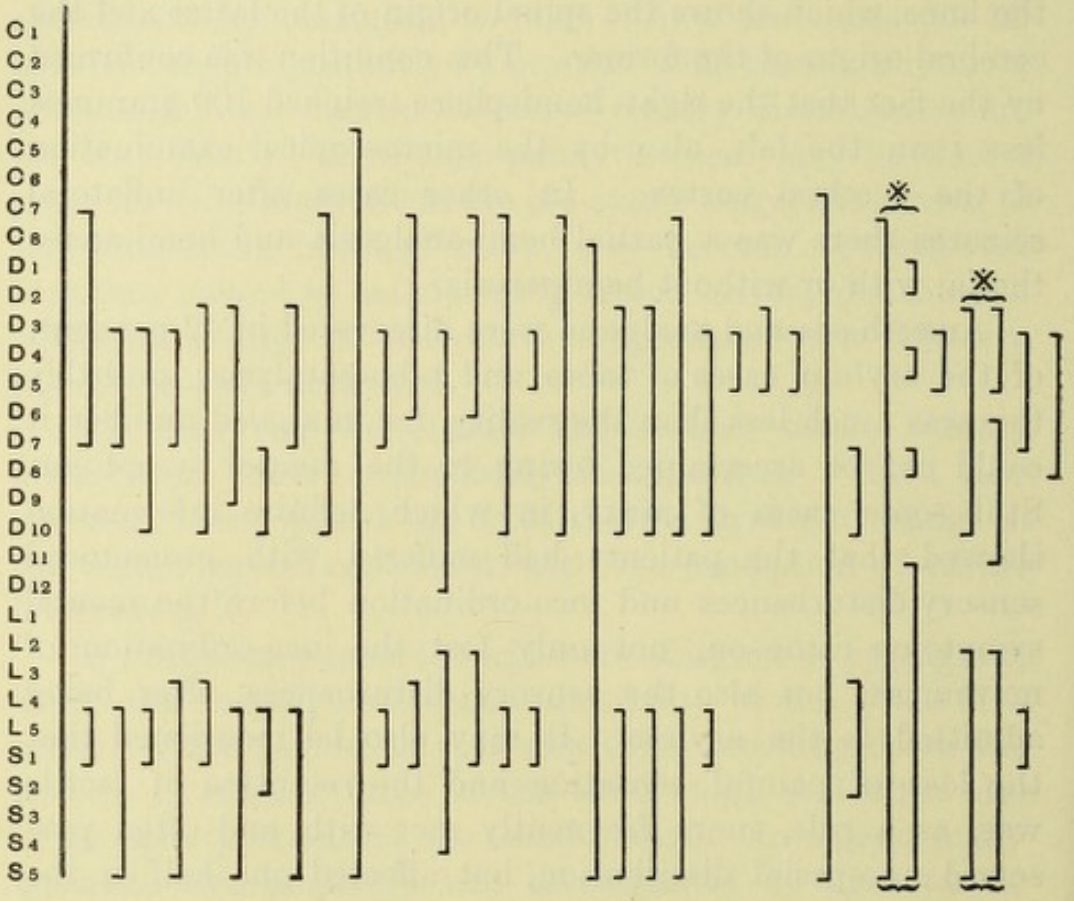


FIG. 3.

Diagram illustrating, approximately, the distribution of cutaneous anæsthesia in the posterior root areas of thirty-two cases of tabes and taboparalysis. Two cases showed unequal affection of the two sides. This diagram shows the regions least affected are the upper cervical, lower dorsal and upper lumbar.

may be anæsthetic. Systematic microscopic examination of the spinal cords and their roots serves to explain these facts. Thus I found in pretty advanced cases of tabes a more complete degenerative fibre-atrophy in the lumbosacral and mid-dorsal regions than in the remaining segments of the cord. Except in arm tabes, the degeneration

of the roots ceased about the eighth or seventh cervical segments. This explains also the fact of the distribution of the anæsthesia shown in the diagram (figs. 3 and 4). Again, on one side we may have roots completely destroyed, while corresponding roots on the other side are only partially destroyed or little affected. The unequal affection of roots will not only account for cases where there is asymmetrical distribution of anæsthesia and anal-

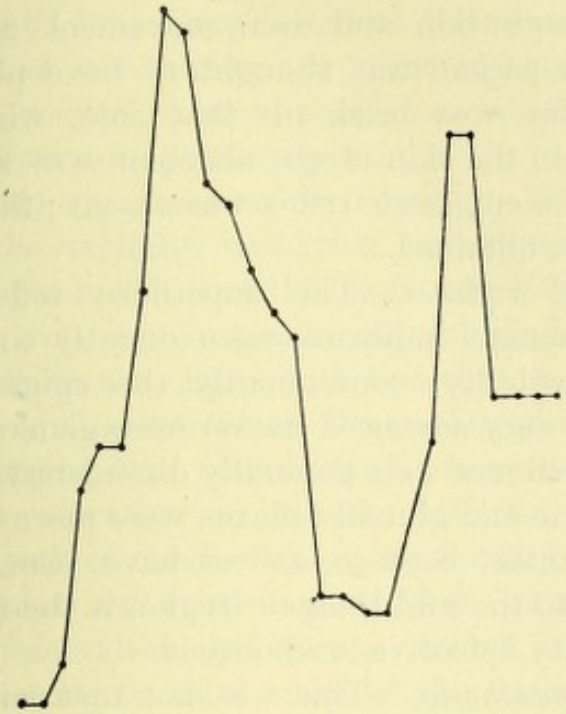


FIG. 4.

This curve is constructed from the previous diagram. The summit of the first elevation corresponds with the fourth dorsal, the summit of the second elevation with the fifth lumbar and first sacral. It is possible that each of these elevations denoting tendency to degeneration of intramedullary portions of posterior roots may be dependent upon some morphological condition, the most likely being a precarious vascular supply; indeed, this is not improbable, for these regions of the cord depend more upon small and variant arteries for their blood supply than the cervical, lower dorsal, and upper lumbar regions.

gesia, but also for hyperæsthetic zones on one side, with anæsthetic on the other; likewise, a widespread distribution of anæsthesia and analgesia, in the midst of which are islands of varying size which are still sensitive to light, tactile sensation, or pricking of pin, may be accounted for by the fact that some of the posterior spinal neurones, with their spinal projections and intraspinal terminals,

are still intact. As the diagram shows, and as the charts in fairly advanced cases (pp. 108-113) of tabes show, there is usually a gap in the regional distribution of anæsthesia over the abdomen and thighs; these skin areas are supplied by the lower dorsal and upper one or two lumbar roots, and it is these roots which microscopic examination show to be less affected. Now this region is usually sensitive to pain and touch, and, indeed, may be hypersensitive. In one case (14) where the skin was hypersensitive on the right side, deep inspiration and even movement caused severe pain, and the patient was thought to have pleurisy. The epigastric reflex was brisk on that side, whereas on the left side, where the skin of the abdomen was analgesic and anæsthetic, the epigastric reflex was absent; the cross reflex was, however, obtained.

Superficial reflexes.—The superficial reflexes, as the previous statement indicates, were directly correlated with the skin sensibility, consequently the epigastric reflexes (excepting in very advanced cases) were generally obtained. The plantar reflexes were generally diminished or abolished; the cremasteric and gluteal reflexes were more often present than the plantar, because, as we have seen, the plantar surface next to the mid-thoracic region is the most likely to have sensibility defective or abolished.

Thermo-anæsthesia.—This was not met with nearly as often as affections of light tactile sensations. Regions which were insensitive to pricking or touching with the tip of the finger appreciated heat and cold; but very frequently the tube containing hot water could not be distinguished from that containing ice-cold water, and in advanced cases both were said to have caused a pricking or burning sensation. One tabo-paralytic, after once being tested with the cold tube, refused to touch it again, as he said it burnt him. Over the abdomen in the hyperæsthetic area, patients were generally hypersensitive to cold. Delay in response was frequent in all forms of sensory disturbance. This was generally in proportion to the intensity and extent of the anæsthesia. In advanced cases also, if the patient felt the stimulus, it was wrongly localised. Occasionally there was

allochiria; in one case, for example, the sensation was referred to the other limb, and in a few instances to a point of the skin above the next joint higher up in the limb. A stimulus which was not at first felt would subsequently provoke a response by repeating it a few times, thus illustrating the effect of summation until the neurone threshold is crossed by the impulse.

Ulnar sensation of Biernatsky was tested in many cases, and it was found that compression of the ulnar nerve at the elbow produced no tingling or pins and needles in the fingers; but in many normal people one cannot sufficiently compress the nerve to produce pain.

Insensibility to pressure of Testicles.—In some cases pressure of the testicles was tried, and it was found that pain was not produced; but this sign, which is undoubtedly a useful one, was not systematically adopted. Sometimes impotence may be associated with this sign.

Vibration of Tuning-fork on Bone.—Dejerine has pointed out that a tuning-fork placed on the bone causes no sensation of its vibration in tabetic patients, indicating a break in the path of conduction of the sensory impulse from bone. This sign was tried in some of the cases which later came under observation, and always verified in patients who were in the second or third stage of the disease (Case 22). Several cases in the pre-ataxic stage did not yield the sign, but I have ascertained in one case that diminished sensibility may exist very early, and be unequal in the two legs.

Visceral Disturbances.

Bladder troubles are among the earliest and most constant symptoms of tabes; they are not severe, and in my experience the patient does not frequently seek advice for this reason. Comparatively few of the cases that came to the hospital sought relief especially for bladder troubles; it is only as the result of inquiry, as a rule, that the patient, in relating his symptoms, mentions difficulties in starting micturition or holding his water. When asked if he has noticed any difficulty with his water, the patient will tell you either that he has a difficulty in starting the stream,

requiring a strong voluntary effort on his part, or that it lacks force, and takes him time to empty his bladder; in the later stages he does not empty the bladder completely, consequently he frequently suffers with residual urine, and catheterisation is necessary. This condition indicates lack of power in the detrusor urinæ, whereas another frequent condition met with, indicates loss of reflex tonus in the sphincter; for a cough, laughing or any cause leading to increased intraabdominal pressure, suffices to cause the escape of a little urine into the urethra, followed by the urgent desire to micturate.

Bladder crises have been described, but they are rare. I have not met with a case. They consist of violent pains which occur in the lower part of the belly radiating to the urethra, and the inner side of the thigh. The patients have an urgent desire to micturate, but are unable; they experience the most severe burning and cutting pains in the urethra, and these may be associated with lancinating pains throughout the lower extremities. These crises may last a few or many hours.

In about *one half* of the cases which I observed there was a history of bladder troubles; this is much too few. The disparity with the results of Leimbach's analysis of Erb's patients (80 per cent. bladder disturbances) may be accounted for by the fact that in out-patient practice in London, where there are so many hospitals, many of the cases are seen only a few times.

In the tabo-paralytic cases, bladder trouble, apart from the incontinence of dementia, was only discovered in 60 per cent, probably again much too few, for examination of the spinal cords in a number of these cases showed that the lumbo-sacral segments of the cord were especially liable to the degenerative atrophy.

Renal crises have also been described. Some of these cases may doubtless be due to the passage of a stone or gravel in a patient suffering with tabes.

Gastric Crises.—In twenty-one patients out of sixty (36 per cent.) gastric crises occurred, and they were not infrequently the earliest symptom of the disease, as the attacks

of pain and vomiting were the sole cause for which the patient sought relief. In one, Case 7, the patient was admitted for intestinal obstruction, and the surgeon was sent for with a view to operation.

Recently I have seen a case in which visceral crises were very severe, and tabic symptoms so ill-defined that although the patient was seen by several distinguished physicians, the true nature of the disease was not discovered, and a surgeon was called in, who performed an abdominal section, and found all the organs normal. He had absent knee-jerks at the time ; it would be interesting to know if he had not Argyll-Robertson pupils and thoracic anæsthesia, as he undoubtedly had when I saw him later. Moreover, he said that before the operation his legs had given way on occasions, and symptoms pointing to rectal crises were recounted. I am informed that the reason why the operation was performed was the existence of pus and blood in the urine on intermittent occasions, plus a large movable kidney. The symptoms seemed also to suggest pyloric disease.

In all cases, as previously stated, the crises were associated with complete or partial anæsthesia in the mid-dorsal region, and often with persistent subjective girdle sensation, but by no means every case of thoracic anæsthesia in this region was associated with gastric crises. This would show that if the crises are due to affection of afferent visceral nerves they are affected independently, and that the relative frequency and early appearance of gastric crises is due to the fact that they or their intraspinal terminations are under the same determining contributory factor to degeneration as the intraspinal terminals of the posterior roots supplying the skin of the mid-thoracic region. One intelligent patient, Case 3, remained in the pre-ataxic stage twenty years, the sole symptoms of the disease of which he was conscious being attacks of severe pain, " sharp " in character, which extended both vertically and transversely between the shoulder blades and over the whole back ; as soon as the pain became acute, vomiting would take place and relief would come. He had no pains in the region of the epigastrium, the attacks of vomiting would last three or four hours.

The attacks of vomiting may be preceded by a pain or a feeling of weight at the epigastrium, in one patient the vomiting was preceded by intense occipital headache, and a feeling of distension of the stomach, and this was followed by lightning pains through the whole body, except the face and the leg which was analgesic and anæsthetic. Severe attacks may last several days, the patient vomiting the contents of the stomach, whatever they may be, and the retching continuing with short intervals, just as in sea-sickness or cerebral tumour, only mucus or watery mucus frequently mixed with bile being voided. In several severe cases, the patients have occasionally vomited coffee ground material, or blood, due probably to rupture of congested vessels. The patients suffering with severe attacks are quite unable to retain food or even water in the stomach, and the pains may be so agonising that they shriek out. The tongue is dry and furred, the lips parched with thirst; worn out with the want of sleep and the distressing symptoms, it is remarkable how soon they recover as soon as the attacks cease. Chemical analysis of the gastric secretion has given variable results, but generally the acidity is diminished.

Occasionally the patients suffered with incomplete gastric crises in which there were only paroxysms of cramp-like pain of the stomach, or only eructations and vomiting without pain.

One patient, Case 57, suffered for years with "rheumatic pains" and "indigestion," for which he was treated at several hospitals without relief. After three years he had delusions of persecution, that unseen agencies turned on electricity and blew up his stomach, and he was admitted to an asylum where his ataxia and progressive dementia were observed. *Post-mortem* examination of the spinal cord showed the typical tabic lesion, and there is no doubt that he suffered with modified gastric crises.

A few cases of gastric crises were occasionally accompanied with frequent purgations. I did not personally meet with any attacks of the intestinal crises which have been described as occurring occasionally; but see Case 46 of tabo-paralysis. They are said to be unaccompanied by pain, apparently suddenly and without cause, frequent watery evacuations of the bowels take place for days, weeks or

months, and then as suddenly cease as they appeared. *Rectal crises*, however, are not so rare; they were met with in 8 per cent. of the cases, and like gastric crises were among the early symptoms. The patients complain of tenesmus, and urgent desire to stool, of severe pain in the back passage like the introduction of a hot iron; sometimes this is accompanied by tenesmus and straining, and it is said evacuations of blood and slime may be passed. Some of these cases may have been institution dysentery. Much more frequently the patients suffer with constipation and difficulty of relieving the bowels without purgatives. Not infrequently they are unable to keep themselves clean. Especially is this the case when they have to start micturition by strong voluntary pressure of the abdominal muscles; fæces are then apt to escape owing to some loss of the reflex tonic contraction of the sphincter ani; moreover, they cannot always tell when defæcation is complete.

In 8 per cent. of the tabo-paralytic cases gastric crises occurred. These visceral disturbances are of great importance, and, in a few instances, seemed to account for the delusions with which the patients suffered, *e.g.*, one patient accused the nurse of having put something chopped up in her milk, and of having scraped her bowels out. Case 31.

Laryngeal Cases.—In only two cases were laryngeal crises met with, and these occurred early in the disease. This symptom was only once (Case 27) met with in the asylum cases, and it is relatively infrequent. The symptoms may in some respects simulate an attack of whooping cough or laryngismus stridulus. One man complained of a sudden spasm of the glottis, with difficulty of breathing, which lasted a few minutes; at the time of the attack he suffered with a burning pain in the larynx and a feeling that he was going to be suffocated. By closing the mouth and breathing only through the nose the attacks were diminished in duration. The attacks are accompanied by stridulous inspiration and followed by short sharp coughs. The crises are not so frequent as visceral crises, and months or years may intervene. The attacks are occasionally very alarming, and may be accompanied by epileptiform seizures and loss

of consciousness. These crises, together with laryngeal paralysis (usually adductor, sometimes one-sided, sometimes bilateral), are considered as evidence of affection of the vagus and spinal accessory rootlets. Sir William Gowers remarks that "in all, if not most, of these cases of adductor spasm there is permanent weakness of the adductors, which, when considerable, constitutes a very grave complication. In rare cases the spasm may proceed to the pharynx, making swallowing impossible, and in one recorded case the spasm extended to the respiratory muscles, causing asphyxia and death." *Bronchial crises* have been described, and in one case the onset of gastric crises was followed by disappearance of this symptom.

Retardation or irregularity of the heart's action have occurred in connection with gastric crises, and precordial pain with anginal attacks have been described (*vide* Case 27).

Genital Organs.—Impotence may be an early or a late symptom. It may be preceded by satyriasis. In some cases impotence is associated with anæsthesia of the external genital organs; in some it is associated with atrophy of the testicles. Diminished sexual power occurred in some of the cases, and absolute loss of sexual power in, at least, 16 per cent.; this, too, may be an early or late symptom. Increased sexual desire is pretty frequent in the early stages of general paralysis and in the tabic form of the disease.

Atrophy of the testicles is of frequent occurrence in general paralysis, and, generally speaking, the testicles lose weight out of proportion to other organs. The satyriasis in a tabo-paralytic may be associated with delusions of extraordinary sexual power; there is often marked desire, but no ability of performance of the sexual act.

Sense of Position of Joints.—The sense of position of joints was tested in thirty cases, and in seven no appreciable affection was shown. All of these seven were early cases, and the diagnosis of tabes was usually based on other evidence than ataxy, viz., Argyll-Robertson pupils, lancinating pains, optic atrophy, visceral crises, and trunk tactile anæsthesia. In all of these seven cases there was no appre-

ciable disturbance in cutaneous sensibility of the limbs. In four very pronounced cases of Charcot's knee-joint, there was no loss of sense of position discovered in the toes; but one of these had impaired cutaneous sensibility to pain and touch in the legs (Cases 11, 5). There was usually a distinct correlation of loss of sense of position of the joints and the degree of ataxy, also between the disturbance of cutaneous sensibility and the joint sensibility. Thus, in one case of arm tabes, there was loss of joint sensibility in the arms, especially the right, in which the disturbance of cutaneous sensibility was most marked. In twenty-one out of thirty cases examined, loss of joint sensibility was found in the toes. In eleven the sense of position in the joints of the whole lower extremity was affected, but nine of these cases were in the third paralytic stage, and there was a correlated loss of sensibility to touch and pain, and in some cases also marked thermo-anæsthesia. In nine out of thirty cases the joint sensibility in the upper limb was affected, and in all cases accompanied by disturbance of cutaneous sensibility, especially of the post-axial border of the limb. In three very advanced and prolonged cases of ataxy, the joints of the whole limbs were affected; in the others it was only the fingers (especially the ring and little fingers) or the fingers and the wrist joints. In one case of fairly complete loss of joint sensation of the upper limb there was only moderate disturbance of cutaneous sensibility. The joint sensibility was lost in the hand on the right side in one case where the loss of cutaneous sensibility was most pronounced. The conclusion arrived at is that loss of sense of position in joints is a very important factor in the production of inco-ordination of movement; it is associated usually, but not necessarily, with loss or disturbance of cutaneous sensibility.

The fact that the toes and fingers are affected first, then the ankle and wrists, and later the joints above, agrees with the distribution of objective sensory disturbances. No doubt the proto-neurons, subserving cutaneous and joint sensibility, are quite independent structures; but, as a rule, they are simultaneously affected. I believe this sense of

position of joints is due to a complex of sensations arising in alterations in *tension of structures about joints*, rather than in the alteration of contact of surfaces such as Prof. James assumes to be the case in judging the sense of position of limbs. It may be pointed out that no one has demonstrated nerve-endings in articular cartilage; there are, however, nerve structures in the synovial membrane and capsule of the joint, in the tendons, muscles, and fibrous structures. The most important nerve-endings are undoubtedly the Pacinian corpuscles, which are especially constructed to be influenced by alterations of tension and pressure in the deeper structures. In support of this argument is the fact that arthropathies (Cases 5, 11) may occur without loss of sense of position and without ataxy, although I have not met with a case in which sense of position of the joint was lost without ataxy.

I have met with several cases of ataxy without loss of sense of position of the joints, even of the toes, and several tabo-paralytics, who were able to give reliable answers, showed the early symptoms of ataxy without loss of joint sensation.

Deep Reflexes.—In seven cases, not tabo-paralytics, nor with cerebral lesions, the knee-jerks were present on both sides. In three the knee-jerk was present on one side and absent on the other; that is to say, absent knee-jerks were found in less than 70 per cent. of my cases when first seen, but then a considerable number of the patients were in the pre-ataxic stage; in fact all the ten cases, except a case of arm tabes, were in the pre-ataxic condition, most of them suffering with optic atrophy or other ocular troubles; two came with gastric crises. In two cases the knee-jerks disappeared first on one side then on the other, after a series of attacks of lightning pains in the legs.

The triceps jerk was not observed in the earlier cases which came under my observation; but it was usually found absent when the knee-jerk was absent, although there might be no other symptoms affecting the arm. In some cases, however, it was present when the knee-jerk was absent; it undoubtedly disappears later than the knee-jerk.

Tonus.—A diminished tonus (as tested by the Fränkel method) was found in all but eight cases, in which the patients were in the pre-ataxic state. In other cases there was a distinct relationship between the degree of hypotonus and the ataxy of the limbs, and if there was a difference in the degree of ataxy of the two limbs it was observed that more marked hypotonus was present in the more ataxic limb.

In the asylum cases the knee-jerks were absent on both sides in 77 per cent., absent on one side in 16 per cent., present on both sides in 7 per cent. As a rule the hypotonus was not so marked as in the tabic cases met with in the hospitals and infirmaries, nor was the ataxy. This is possibly due to the cerebral disease withdrawing the normal inhibitory influence. With the onset of the mental symptoms, the inco-ordination of gait and station and muscular hypotonus underwent improvement, and the symptoms, in some cases, apparently so far disappeared that one was not able to prognosticate the extensive degeneration of the posterior columns which I found in many of them.

If, however, the patient has advanced well into the second stage of tabes, the onset of the mental symptoms is only accompanied by a less degree of ataxy, and the characteristic inco-ordination in gait and station in great measure persists, so that one is easily able to pick him out from a number of cases of general paralysis as a tabo-paralytic. As the brain becomes affected the ataxic gait often gives place to a shambling or shuffling gait, and I have been surprised what an extensive amount of degeneration of the posterior roots and posterior column of the spinal cord may be found in such cases. Very frequently (especially if the patient has had epileptiform seizures) this is combined with very extensive crossed pyramidal degeneration. So that although one was unable to obtain the knee-jerks on either side, yet, on the side of the fits and hemiparesis, Babinski's sign was obtained, *vide* Case 54.

In one case (44), however, the knee-jerk returned on the side upon which the fits occurred.

Romberg's symptom, so frequently a constant and

prominent symptom in ordinary tabes, may be only slightly obvious in asylum cases.

25 % tabo-paralytics, slight ataxia only discoverable by careful examination. Romberg's symptom absent.
30 % ,, moderate ataxia observable in gait and station. Romberg's symptom present.
15 % ,, marked ataxia advanced second and third stage.
30 % pre-ataxic tabo-paralytics, no ataxy discoverable; diagnosis made by symptoms, lightning pains, optic atrophy and absent knee-jerks. Very frequently subsequent examination of the spinal cord by careful microscopical examination has demonstrated the correctness of the diagnosis.

Romberg's symptom was very marked in only 15 per cent. of the cases, moderately in 26 per cent. and slightly in 7 per cent.

Section III.

Affections of the cranial nerves are less frequently met with; it is not common to find the olfactory nerve affected in tabes, but occasionally, as my cases have shown, loss or imperfection of smell and the taste for flavours has occurred. Sometimes, as Case 10 shows, this was preceded by a curious and continuous odour in the nostril, like a drain; it is sometimes likened to rotten fish, phosphorus or sulphur, and paroxysmal attacks of this perverted olfactory sense may precede the complete loss of smell. These attacks, when they occur, are of the nature of crises and may last hours, days or weeks; it is of importance to bear this in mind in connection with the subjective attitude of an insane tabetic patient, or a tabo-paralytic, to these abnormal sensations; unable to explain their presence, he might put an insane interpretation on them, thinking that his food was poisoned, or that foul odours by unseen agencies were working upon him. Although loss of smell and the taste for flavours is uncommon in tabes, it is relatively frequent in tabo-paralysis and general paralysis, and this accords with the anatomical results found *post*

mortem, for the orbital surface of the brain and the tip of the temporal lobe in which it is presumed the sense of smell and taste is located, are very generally the seat of extensive atrophy and thickening of the membranes.

Sometimes the patients are said to lose sensibility of the mucous membrane of the nose, and no longer sneeze when it is irritated, or they may be subjects of paroxysmal attacks of sneezing and tickling of the nasal mucous membrane. This is due to affection of the nasal branch of the fifth nerve, and is rare. Other affections of branches of the fifth nerve are pains, anæsthesia and paræsthesia in various regions of its distribution. Besides these disturbances of sensibility, there may be various so-called trophic disturbances, such as a rapid falling out of the teeth, and absorption of the alveolus of the jaw. Several of the cases showed this, but in some instances (Cases 2, 15, 17) it may have been due to the mercury given in the treatment of syphilis. The sensory disturbances of the fifth are sometimes manifest, and sometimes so trifling as to be unobservable; there does not appear to be any relationship of the teeth dropping out and absorption of the jaw to anæsthesia of structure supplied by the fifth. This is in accordance with what we know of joint and bone diseases, which are frequently very early symptoms, and are in no way related to the sensory disturbances of the skin; the teeth fall out without any pain being experienced.

Other rare conditions due to affection of the trigeminus have been described, namely, ophthalmia neuro-paralytica and corneal ulcer. Both corneal ulcer and tabic ulcer of the mouth are analogous to the perforating ulcer of the foot. Again, sialorrhœa, which occasionally occurs, generally in paroxysmal attacks, has been attributed to affection of the trigeminus.

Disturbances of taste, due to affection of the glosso-pharyngeal nerve, are very rare; cases do occur occasionally in the form known as medullary tabes (see Case 62).

Auditory nerve.—The result of affection of this nerve as in other cases may be irritative or paralytic. Paroxysmal attacks of noises in the ear like rushing water, bells or

shrill whistling sounds, or even musical sounds may occur (Cases 62, 24). This, no doubt, is due to the affection of the neurons supplying the labyrinth ; but the neurons supplying the semi-circular canals may also occasionally be affected, and give rise to symptoms like Menière's disease, namely, attacks of giddiness or loss of balance. But it is difficult to determine whether this is not really due, in the absence of other signs of affection of the auditory nerve, to migrainous attacks or slight attacks of *petit mal*, or the giddiness of congestive attacks occurring in patients who are in the initial stage of progressive paralysis. Occasionally, as Case 62 shows, absolute deafness may occur.

Diseases of the Bones and Joints.

Arthropathy and bone affections occurred relatively frequently in my cases of tabes. They were met with oftener in women than men, and not infrequently were associated with gastric crises (Cases 5, 17). Spontaneous dislocation or fracture was often the cause of the patient seeking medical or surgical advice. Joint affections and spontaneous fractures were met with not only in hospital, but also asylum cases. It is of considerable importance to bear this in mind, otherwise officials and attendants might be charged unjustly with neglect or ill-treatment, when in reality a very trivial accidental injury or no real injury, merely turning the patient in bed, might be followed by spontaneous dislocation or fracture in asylum cases of tabes or tabo-paralysis. Not long since I saw a fracture of the thigh in a young man suffering with juvenile tabo-paralysis ; the spinal disease was not discovered during life, but microscopical examination of the cord showed the characteristic lesion. It would have been easy to have cast blame in such a case as this, for the immediate cause of death was due to infective suppuration around the seat of fracture. Such fractures may occur apparently spontaneously or as a result of a very slight injury, or even a false step ; they are usually quite painless, and one patient told me she did not know her leg was broken until she saw

the ends of the bone sticking up (Case 71). A considerable number of women in workhouse infirmaries lie there incapacitated for numbers of years with tabic joints. One of the earliest cases reported by Dr. Buzzard is still in the St. Pancras Workhouse, where she has been nearly thirty years; the gastric crises and lightning pains, from which at one time she suffered excruciating agony, have latterly almost ceased. The joint affection may, as in Case 5, be the sole symptom causing the patient any trouble; for of course the physical signs, namely, the existence of the Argyll-Robertson pupils and absent knee-jerks, would be unknown to the patient, and if there were lightning pains, they would be put down to rheumatism. There is no doubt that, as in rheumatoid arthritis, hard occupations involving much use of joints predispose to the disease. Case 5, a stonemason, in whom a large painless swelling of the right shoulder-joint was the first symptom, is an illustration, for he used this arm for his hammer. Again, a carpet-planner, who knelt all day, had an enormous painless swelling of both knee-joints. The poor women I met with in the infirmaries suffering with Charcot's knee-joints nearly always gave a history of being widows who had to earn their living by charing and scrubbing, or by using the sewing-machine. The two cases of tabic foot met with, were men who had been in the army, and subsequently worked at a forge, and were, therefore, standing all day at their occupation. In the cases which came under my notice of joint affection there was usually a history of a trivial injury such as a knock, a false step going downstairs or from the kerb, or such like. The swelling was not invariably painless, and it appeared to me sometimes difficult to differentiate some of the cases from rheumatoid arthritis occurring in a person suffering with tabes or tabo-paralysis, had not rapid effusion into the joint indicated a true tabic arthropathy.

Symptoms.—A masterly description by Charcot of the tabic arthropathies has led to the affection being named after him. He pointed out that without any sufficient cause a joint would suddenly swell up, owing to a serous exudation into it and possibly into its surrounding tissues. This

swelling is unaccompanied by either pain or fever. In the favourable form the swelling may disappear after a time, and a return to the normal condition take place, or severe destruction of the joint may occur accompanied by crepitus, wearing away of the articular surfaces and even the bone, causing dislocations and luxations. The knee is by far the most frequently affected, after this the hip, shoulder, elbow, wrists, more rarely the ankle, vertebral, and finger joints. Marie states that from 4 to 5 per cent. suffer from joint affections. Among my cases there were 10 per cent. The joints were examined in two of the cases; one was hardly characteristic, for a sinus existed with dead bone and continuous purulent discharge; the leg was amputated by Mr. Walsham, the capsule of the joint was greatly thickened and dilated, there were numerous osteophytes, and the cartilage was ulcerated but not entirely destroyed; there was an osteo-porosis of the bones; some nerves were dissected out that were proceeding to the peroneus longus muscle, and separated from the surrounding tissues away from contact with the suppurative process. A small piece was cut off and teased, the fibres were found (after suitable staining) to be undergoing an atrophic degenerative process (Case 30), but the appearances are not unlike those described by Marinesco after amputation; and seeing that the leg was extremely wasted and the joint so disorganised as to be quite useless—hence the amputation—it may be asserted the condition was an effect of the joint disease. In a case of tabetic general paralysis the knee-joints presented an appearance in some respects resembling rheumatoid arthritis both during life and *post mortem*. The capsules and synovial membrane were thickened, contained excess of synovial fluid, and the cartilages were ulcerated (*vide* fig. 5). The patient was too demented to say whether it was painless or not. This was a case of very advanced tabes, and there was atrophy and degeneration of the peripheral nerves and the small nerve fasciculi of the skin.

In advanced cases the joint surfaces with the cartilages may quite disappear, and the bony structure in the joint may be destroyed and appear as eroded away. Proceeding

pari passu with the atrophy there may be numbers of outgrowths in the capsule and synovial membrane. Chemical examination shows that the bones are deficient in mineral matter, especially phosphates. The Haversian canals are dilated irregularly and filled with fat. According as the process affects the epiphysis or the diaphysis an arthropathy or spontaneous fracture occurs, sometimes both (Case 71). Similar joint affections may occur in syringomyelia.

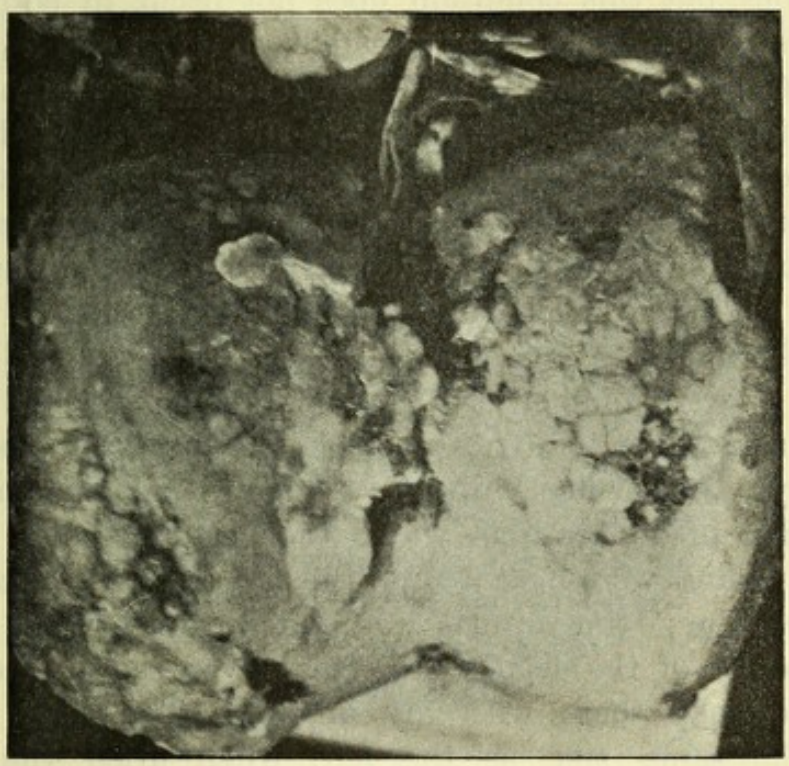


FIG. 5.

Photograph of the articular surface of the femur from a case of arthropathy in a typical tabo-paralytic female with all the clinical symptoms of the disease. Microscopic examination of the brain and spinal cord showed characteristic lesions.

A peculiar malformation arises when the joints of the tarsus are affected; on the back of the foot and in the middle of the sole there arises a hard prominence, the foot is flattened and shortened and the bones of the tarsus are, as it were, driven into one another. Crepitus is obtained on movement, but no pain produced. The ligaments of the joints may be destroyed; several such were met with

both in asylum and hospital cases. Tendinous tissue may probably undergo similar atrophic changes, for sudden painless rupture of a tendon which does not tend to heal may take place.

Whether the bone and joint disease of tabes is dependent upon a definite lesion of the nervous system is still a moot point. Charcot believed at first that it was due to a lesion of the anterior horns. Other authorities attribute it to a disease of the peripheral nerves, especially of those supplying the bones, but this condition does not occur in peripheral neuritis. The loss of bone sensibility may, however, be an early sign in tabes. I recently examined a patient who presented very little ataxy and yet there was very distinct evidence of diminished bone sensibility by Egger's test, with the vibrating tuning fork. Recently Marinesco has attributed it to anæsthesia and absence of reflex regulation of the blood supply; it may also be due to the direct action of toxins upon the nutrition of the bones.

In a large number of experiments which I made many years ago of section of anterior and posterior roots in monkeys, I never met with any change in the bone with the exception of one case in which the posterior roots of the cauda equina were ligatured on the right side so as to cause degeneration; the animal was killed three weeks later. The bones of the leg on this side were so brittle that I could snap them with my fingers. The right femur was the same size as the left, when dried the right weighed 7·5 grams, the left 12·5 grams. The right os innominatum was the same size as left, when dried the right weighed 5·5 grams, the left 7·7 grams. The tibiæ were also brittle, but were used for microscopical purposes. After softening in chromic and picric acids transverse sections were cut by the freezing microtome, stained with logwood and eosin, and mounted in Farrant's solution.

There was a naked eye osteo-porosis on the right side. The periosteum was apparently normal. Examined microscopically there was a marked dilatation of the Haversian canals, and owing to the absorption of the intervening bone, coalescence had taken place with the formation of irregular

shaped channels, which contained blood-vessels surrounded by a reticulum of connective tissue in which were seen numerous osteoclasts ; these large cells were disposed usually at the periphery lying in little scalloped out places which they filled and which they apparently had caused, by absorption of the osseous substance (*vide* photomicrograph, fig. 6).

Bayliss and Starling have shown that stimulation of the lumbo-sacral posterior roots causes dilatation of the vessels of the lower limb ; therefore vaso-dilator excitation may have

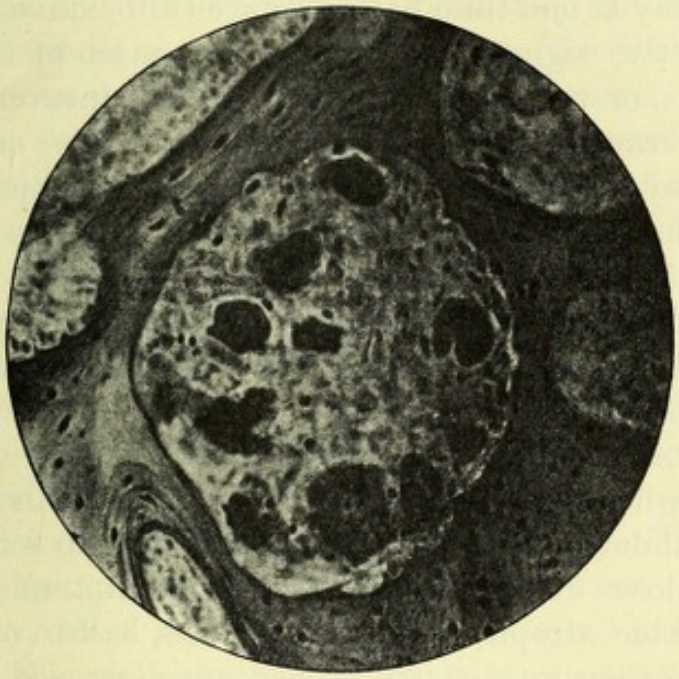


FIG. 6.

Photomicrograph of section of tibia softened in a mixture of picric, nitric, and chromic acids, stained with alum hæmatoxylin and mounted in Farrant's solution.
Magnification 300 diameter.

been set up by the ligature and caused dilatation of the vessels of the lower limb including those of the bones, with consequent absorption and diminution of the mineral matter ; for although, as we have seen, the bones were equal in size, there was a marked difference in the weight, and microscopically it was found that this was due to absorption o the osseous matter around the Haversian canals.

Motor Disturbances.

The cases may be classified clinically as follows, according to their motor disturbances :

(1) Motor disturbance slight or not discoverable. Preataxic condition, disease spinal :—Loss of deep reflexes on one side or both sides, slight hypotonus. No subjective motor trouble. Objective signs only discovered by expert examination. Other signs and symptoms lead to diagnosis.

(2) Preataxic condition simulated, spinal combined with cerebral or optic disease :—In such cases the spinal disease may at one time have produced subjective symptoms and objective signs of ataxy, and the onset of the mental symptoms or the optic atrophy with blindness masks the same, or causes them to disappear partially or entirely. There may be loss of knee-jerks, or the knee-jerk may be lost on one side only, or it may return on one side after a series of unilateral fits. Cases 41, 44, 65 show that these patients have suffered with extensive degeneration of exogenous fibres sufficient to produce fairly marked ataxy. Most common in asylums.

(3) Ataxy of first stage, subjective difficulty only in the dark, or with eyes shut discoverable by examination, same as (2) without mental affection :—Loss of deep reflexes, well marked loss of tonus, Romberg's symptom moderate. Considerable atrophy of posterior roots, half to one-third of fibres and their intra-spinal projections destroyed.

(4) Ataxy of second stage, obvious ataxic gait, subjective difficulty in daylight necessitating support :—Nearly complete atrophy of the lumbo-sacral root-fibres, considerable atrophy of endogenous systems of fibres, especially descending comma, septo-marginal, oval area, and Phillippe's triangle. Case 39.

(5) Paralytic bed-ridden stage, frequently accompanied by deformity and great muscular wasting, especially of certain groups of muscles, which may in rare instances undergo degenerative atrophy. Cases 28, 31.

The fuller consideration of the motor disturbances is discussed in the pathological section, chapter "Incoordination and its Pathology."

Cerebral Symptoms.

I have already alluded to the frequency of optic atrophy in the cases of tabes and tabo-paralysis which have come under my observation. I will now refer to other cerebral symptoms. Mental symptoms occurred in about 10 per cent. of the cases seen outside of the asylums; some few of these were pronounced general paralytics of the tabetic type, others were tabetics with symptoms indicating early organic affection of the brain, and the remainder were cases of tabes dorsalis with associated insanity. Some of the cases seen in the hospitals and infirmaries afterwards died in one of the asylums at a near or remote period from the time I saw them. One case in particular, J. W., attended my out-patients for several years, affected with progressive optic atrophy, limitation of the field of vision, and early signs and symptoms of cord affection. I lost sight of him for some years, but found him suffering with advanced general paralysis in Claybury Asylum, and he died there. The same types of tabo-paralysis were met with in the asylums, the only difference as a whole between the cases was what we would expect: in the former the cord symptoms predominated, in the latter the mental symptoms. It was, however, a well-established fact that some patients who had been observed outside of asylums as cases of loco-motor ataxy, after admission to the asylums, and there-fore after the development of well-pronounced mental symptoms, without treatment of any kind, became less ataxic in their movements, the gait becoming altered. As in general paralysis so in tabo-paralysis, the onset of cerebral symptoms may be sudden, and due to seizures which are recognised as congestive, apoplectiform, epileptiform, and migrainous. The seizures may lead to a disturbance of consciousness, or partial or complete loss of consciousness. Thus a patient may be suffering with discovered or undis-covered objective or subjective signs and symptoms of tabes dorsalis, when suddenly, or comparatively suddenly, he is seized with some kind of fit. There may be a temporary disturbance of consciousness, a sudden feeling of giddiness, associated with frontal headache like migraine. Generally

such an attack is due to circulatory disturbances of the brain, but it is conceivable in rare cases of tabes; frequent migrainous attacks may be due to disturbances of the sensory impressions from the semi-circular canals. These attacks often escape observation, until one more severe necessitates a doctor's advice, then the true nature of the disease may be discovered by the examination of the pupils and the condition of the knee-jerks and other signs of tabes or general paralysis. This will lead to further inquiries of the patient for such early subjective symptoms as lightning pains. Often, however, the patients disregard these slight attacks, and such cases do not come under observation until more serious brain symptoms develop. Some of my cases show that a history obtained from the wife or friends may give evidence of such attacks occurring years before the patient suffers with mental symptoms severe enough to cause his admission to the asylum. Epileptiform seizures may be the first evidence of cerebral affection, and may precede or succeed spinal symptoms. Cases 36, 52, 53.

I have frequently found, by questioning the wife, that her husband, previous to his coming to the asylum or the hospital, had a fit with convulsive spasms affecting one side. He had recovered from this, and was comparatively none the worse for it. In 30 per cent. of sixty cases of tabo-paralysis the patient suffered with numerous epileptiform seizures. In 34 per cent. there was a history of one or more fits, but seizures did not form a prominent symptom of the disease. In the remaining 36 per cent. no fits were observed by the friends or attendants during the whole course of the disease. Epileptiform seizures in the case of well-marked tabes, where there is presumably well-marked degeneration of the posterior roots, had not been notably characterised by inco-ordination in the convulsive spasms. This is a matter of some interest and will be discussed later. The epileptiform seizures may be slight, limited to twitching of one side of the face or to a limb, or they may extend to the whole of one side of the body, or they may become bi-lateral, and the muscular spasm so severe and general as to cause opisthotonus or emprosthotonus. The convulsive seizures

in Case 46 were extremely interesting, for this man was in an advanced stage of locomotor ataxy. Loss of consciousness may be partial or complete, and there is a correlation between the severity and the extent of distribution of the muscular spasm and the depth of unconsciousness; as a rule, when the convulsions are limited to one side, the loss of consciousness is only partial; the pupil is usually larger on the opposite side to that of the seizures; the face is congested, the skin perspiring freely and its surface temperature raised from ·5 to 1·5 degrees Fahr. on the affected side.

When extensive convulsions occur continuing for several days (a condition of *status epilepticus*), there may be high fever, even hyper-pyrexia, which may cause death. Such severe attacks, however, are associated not infrequently with the onset of an acute disease such as pneumonia and dysentery, which kills the patient; and if the bowel has not been examined, and the lungs be only in the first congested stage of pneumonia, the patient's death is attributed to the seizures; whereas really the seizures have been excited by the toxic condition of the blood, and have only accelerated death. The same applies to epilepsy. After the fits have ceased, the patient recovers rapidly, and his mental condition may even appear to have improved. This is not really the case; the functional disturbance of the whole brain caused by the congestion has passed off, and his mind is therefore less affected, but the result of the fits is usually tissue-destruction. In my opinion, the fits are often the expression of an increased irritability preceding decay or even death of the psycho-motor neurons of the cerebral cortex. The degeneration of the pyramidal fibres of the spinal cord is most abundant on that side on which the fits occurred, if they are unilateral; when the fits are bilateral, the degeneration is proportionately great on both sides. My observations would show that acute death of the cells is due in great measure to vascular inflammatory disturbances, but degeneration of the pyramidal fibre-systems, especially those fibres which come from the leg area, may occur independently of any fits, and, as I shall show later, are due to degenerative atrophy commencing in the collaterals and

terminal portions of the fibres, as in idiopathic lateral sclerosis and amyotrophic lateral sclerosis. In some cases the changes are obviously vascular and due to arterio-sclerosis affecting the small vessels; as a rule, however, there are not sufficient gross changes in the arteries to account for the wasting of the brain. In Case 45 the right hemisphere was particularly affected, it weighed 100 grammes less than the left. Although there was such tremendous destruction of the right hemisphere and very considerable destruction of the left, the dementia was comparatively slight; so much so that the attendants did not think that he could be a general paralytic. His speech, moreover, was but slightly affected. He suffered with left-sided hemi-paresis and well-marked hemi-anæsthesia and hemianopsia. The cause of this is discussed in Case 48, where there are well pronounced left sided seizures, no hemi-anæsthesia resulted. There was naked-eye degeneration of the pyramidal system of fibres on the left side of the cord, as in Case 45; but it did not extend to the pyramids in the medulla, and there was no gross lesion of the central convolutions or occipital lobes. As a rule, after seizures affecting one side, say the right, there is temporary hemiplegia, or hemiparesis, or temporary aphasia, and there may be hemianæsthesia. Usually with each series of seizures the speech affection characteristic of general paralysis becomes more marked.

Apoplectiform Seizures. — These may be manifested in different degrees of severity, by slight fainting fits, brief lapses of consciousness described by the friends as "a look of bewilderment," perhaps followed by a transitory aphasia, word deafness, word blindness, or verbal amnesia, or the patient is unable to express general ideas correctly, and the words that he utters have no logical sequence, or may be even meaningless gabble. Such conditions of speech defect may, or may not, be accompanied by transitory hemiparesis, monoparesis, or facial paresis (*vide* Cases 54, 56). Such seizures may precede by months or years all other symptoms. Transitory defects of speech lasting a few hours, a few days, or even a few weeks, are particularly suggestive of two

diseases—syphilitic endarteritis and general paralysis. As a history of syphilis and signs of it will in all probability be discovered in either case, and whereas the one is capable at least of partial, if not of complete recovery, and the other, general paralysis (a progressive degenerative disease), is absolutely fatal, it is of great importance in the treatment and prognosis to determine from which disease the patient is suffering. The presence of Argyll-Robertson pupils is in favour of general paralysis, for in syphilitic brain disease it is much more common to find the pupils inactive both to light and accommodation. Mental symptoms characteristic of general paralysis may not be present. An hereditary history of insanity would favour general paralysis, as, too, any of the symptoms of tabes dorsalis (see Cases 53, 54, 56). Very occasionally, as in tabes, syphilitic endarteritis may co-exist with the primary degenerative disease.

Mental Symptoms.—Our psychical condition, particularly that relating to the feelings and emotions, oscillates without ceasing about a mean point of equilibrium, and the extent of oscillation within the limits of health is dependent upon individual temperament and the circumstances of environment. Difficult as it is to fix the normal in the physical organism, how much more so in the psychical?

Above and below the mean point of equilibrium are grades of pleasure and of pain, which are reflected to the muscles of expression, causing synergic alterations in their minute tensions. Every passing feeling is thus reflected, so "that there is no art to find the mind's construction in the face."

Only those who have had the opportunity of observing the feelings, emotions, intellect and actions of the individual closely, and for some considerable time, can correctly appreciate a change of character, of volition, of intelligence, and thus gauge the earliest signs of the insidious and progressive mental change which usually precedes the more obvious and obtrusive symptoms and signs of brain degeneration. Long before the patient is admitted to the asylum, symptoms indicating a change of character may have been noticed by the wife, relatives, or associates in business. If

the man is married, the wife may tell you that months or years before she had become aware of his being afflicted with insanity, she had noticed a change in his disposition. Previously affectionate and kind, of an equable temperament and happy disposition, he has become subject to fits of temper; or sullen, morose, depressed, and often neglectful of her and the children. Or she has noticed that he worries over the least trifle; accustomed to act independently, confident in his own judgment, he loses all self-reliance, and worries her over the smallest details regarding his actions. He may be restless and excitable, or sullen and moping, and he has become a man of varied moods, and unable to fix his attention on any subject for any time. Or the history sometimes points to the patient having been filled with morbid suspicions, which later become fixed delusions of persecution, causing him perhaps to get up in the night to look for burglars, and to hide his valuables. These delusions and fits of temper may impel him to threaten or even attempt suicide or homicide, and she may be afraid of her life and that of her children. Again, the husband who always brought his money home at the week end, may have neglected to do so, and have become extravagant, boastful, and addicted to drink. Other instances occur of quiet, sedate and staid individuals exhibiting an antithesis of character, giving way to amusement, gambling, immorality, vice and intemperance. Sometimes the prodromal period may be marked by flashes of genius or exceptional brilliancy of artistic or intellectual power. There is nearly always, however, a failure of concentration and steadfastness of purpose to carry the schemes to fruition. The wife may have noticed that the patient has become careless about his personal appearance, untidy in his dress, forgetful of his business or his home affairs, especially relating to events that have recently happened, his mind being absorbed by ambitious schemes which come to naught, speculation, gambling or betting. These prodromal signs of a disordered mind may precede or be associated with early or well-defined symptoms of cord affection. The brain would show at this stage only slight macroscopic change, but definite microscopic changes.

Not infrequently tabo-paralytics become lustful, and their attempts to satisfy their increased sexual desires leads them to adultery and immorality, which may get them into trouble in the police-courts, and their disease may then be discovered. Too often there is a history of the home being broken up, and the wife and children left destitute, showing the necessity of an early recognition of this disease. A condition of satyriasis is usually followed by impotence, and the history related to me by several patients, but which I have not included in the notes of the cases, shows that sexual perversions may arise in consequence. Satyriasis is not infrequently an early symptom of tabes, and when this is combined with mental affection, it may be associated with delusions of extraordinary sexual power, which remain as a fixed idea even when impotence has supervened.

The delusions may take another form; the patient becomes suspicious of his wife's fidelity, and on this account he may become dangerous to her and others. The most common mental affection which brings a tabetic or tabo-paralytic into the asylums is "acute mania," of which we may consider that there are four types.

(1) Acute mania from which the patient completely recovers. This form is generally due to a combination of several of the following factors:—(*a*) toxins from without; usually alcohol; (*b*) toxins from within; (*c*) head injury, business failure, mental shock, worry and anxiety owing to pain suffered, sleeplessness, knowledge of suffering from an incurable disease, onset of optic atrophy ending in blindness, impotence, distress and pain occasioned by visceral crises; (*d*) inherited psychopathic temperament. (Cases 28, 29, 30.)

(2) Acute mania from which the patient does not recover, in which the brain affection becomes chronic and associated with delusions, hallucinations, and illusions, accompanied by dementia, which, however, is usually non-progressive, caused by the same factors as above; but frequently there is a marked hereditary history of insanity in the family (Case 25).

(3) Acute mania becoming sub-acute, then subsiding

completely, associated with dementia which is non-progressive; often a great mental improvement takes place, leaving only traces of intellectual enfeeblement. Such cases are usually tabetics who either in the earlier or later stages of the disease have taken to drink. After admission to the asylum the poison no longer acts upon the nervous system, and the dementia which persists is proportional to the organic destruction of the cerebral cortex by the alcohol (Cases 70, 71). It is extremely difficult to decide whether or not these cases are tabo-paralysis, in which the disease-process is arrested by the removal of exciting factors. I have seen a few cases of absolutely certain general paralysis arrested in progress for years. The patients were even discharged as cured (*sic*); within a few months or less they are back at the infirmary to be re-certified, and sent back to an asylum, where they die within a short time. Presumably they have been unable to stand the mental stress outside the institution, and should not have been discharged.

Many instances of the almost immediate effects of mental shock in inducing an acute exacerbation of the disease have come to my notice. The cause is sometimes avoidable—*e.g.*, a quiet melancholic general paralytic developed acute homicidal and suicidal mania the same night after hearing the fatal nature of his disease discussed by the physician in a ward. A patient who had so far recovered that his discharge from an asylum had been considered advisable, was told by a friend visiting him that he was suffering from general paralysis; this preyed upon his mind, and the disease-process, which was only smouldering, was fanned into flames, and within three months he died from the disease. The same applies to tabes dorsalis, the disease progresses when the patient is subjected to bodily stress, or gives himself up to sexual excitement, drink, and debauchery.

(4) Mania, frequently with grandiose delusions typical of general paralysis, accompanied by symptoms and physical signs of tabes (more or less defined according to the stage of the cord affection), associated with dementia, which, after the subsidence of the mania, is found to be progressive, and is usually accompanied by characteristic defects of speech, verbal and written. The acute mania from which the

patient suffers may be partly due to alcohol, partly autotoxic in origin.

As in general paralysis, cases of tabo-paralysis or tabes may be accompanied by various degrees of mental depression, with delusions of persecution. These may be cases of tabes with insanity, or tabo-paralysis of the melancholic type.

Hallucinations are of great importance because they exercise a powerful influence, even more powerful than perceptions, on the intellect and volition of the patient. Many cases of so-called hallucinations are rather of the nature of pseudo-hallucinations or illusions, for they are excited by peripheral irritation. I have already referred to those relating to the skin and viscera in tabo-paralysis. In many of those cases definite lesions were found *post mortem* to account for the symptoms manifested during life. In cases where there has been almost complete destruction of the posterior roots, there have still been the lightning pains and visceral crises which have been insanely interpreted. Many of the tabetic cases of very old standing still suffer with the lightning pains and visceral crises. All the while there are any rootlets left undestroyed by the disease, pains may occur and radiate all through the sentient grey matter, each decaying fibre serving as a fulminating agent. Among the many cases of optic atrophy leading to partial or complete blindness were a number who suffered with visual hallucinations—viz.: Cases 59, 64, 70. Complex visual hallucinations are usually of human forms or of animals—rats, cats, mice, lions, tigers, snakes; the human forms are generally policemen, burglars, dead relatives, or people who were supposed enemies; and like auditory hallucinations they would tend to engender and aggravate delusions of persecution. It is remarkable that people who are quite blind should thus suffer from visual hallucinations, but it accords with other facts. Uhtoff, in a recent monograph, has recorded a case of tabic optic atrophy with visual hallucinations, and he refers to the fact that numerous cases have been recorded of blindness with visual hallucinations due to various causes. Some of my patients were not, however, absolutely blind, and the

hallucinations may have been caused by the irritation of the progressive death of the fibres. One case in particular which, however, was a general paralytic is of very considerable interest in this respect: the patient was admitted with acute *mania a potu*, placed in a padded room, and while there he was the subject of visual hallucinations. When I first saw him he said black devils came and perched on his nose and put stinking things up his nostrils and nasty things in his mouth. I found he had Argyll-Robertson pupils and I expressed the opinion that he had general paralysis as well. Examination of the eyes ophthalmoscopically showed numerous hæmorrhages around the disc, and chorio-retinitis. A fortnight later when I saw him, and, presumably, the effects of the acute alcoholism had passed off, the hallucinations had changed. He now said angels came and moistened his lips with sweet things and exhaled sweet perfumes up his nostrils. He exhibited many other signs of grandiose ideas. The retinæ were examined after death and the observations during life confirmed; likewise the degeneration of the optic nerves was found microscopically, *vide* photomicrograph (fig. 7). It must not be supposed, however, that the majority of cases of visual hallucinations are due to peripheral irritation caused by disease of the retina or optic nerves; in fact they are more likely to occur from a morbid condition (functional or organic) of the higher ideation centres, presumably situated in the angular gyri. Uhtoff has described, and Henschen has collected, a number of cases of hemianopsy due to destruction of an occipital lobe or the radiating fibres, and in these cases visual hallucinations have occurred on the side upon which the hemianopsy was. The only case of hemianopsy that occurred among my cases (Case 45) did not suffer with hallucinations of vision; therefore, destructive softening of the occipital lobe does not necessarily, even in a taboparalytic, produce this condition; it may be presumed that in those cases where hallucinations occurred, there existed a morbid functional or organic disease of the corresponding ideation centre of vision, which was excited by the irritation of its associated diseased perceptive centre, or the peripheral

structures (retinæ and optic nerves) connected therewith, *vide* Case 26. This morbid condition is due either to inherent instability or general toxæmia, the combination of factors being sufficient to produce the hallucinations.

The visual hallucinations were generally associated, like the auditory, with delusions of persecution. In one case a condition of *macropsy* occurred. The patient, who was a

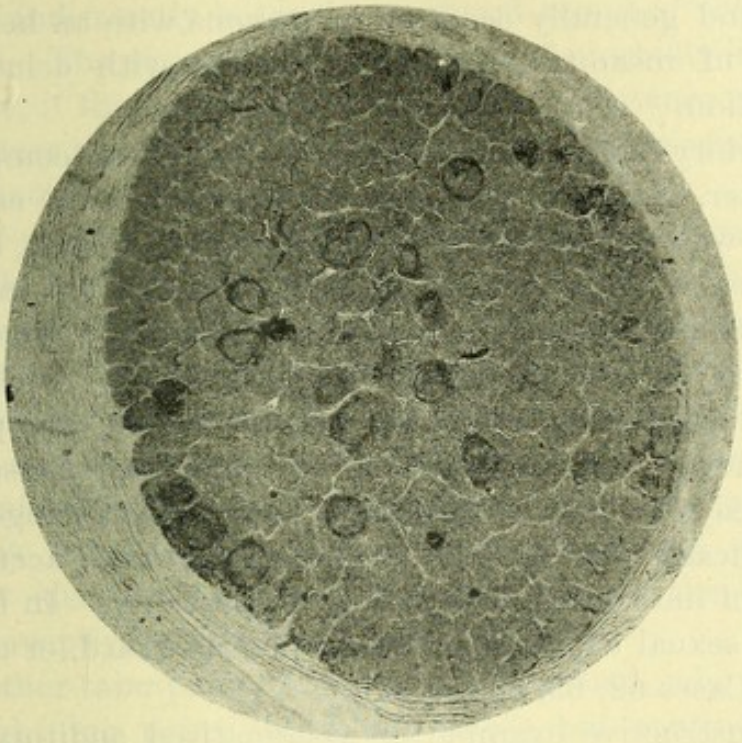


FIG. 7.

Photomicrograph of a section of the optic nerve, stained by Marchi method. The black circles indicate bundles of fibres containing abundant degenerated fibres; it will be observed that not more than 15 per cent. are affected, and these doubtless corresponded to the paletus of chorio retinitis observed during life.

Magnification 25 diameters.

very intelligent man, said everything appeared much larger than natural. Another patient, suffering with a curious complex of symptoms (Case 72), complained of things moving which were stationary. He had very sluggish re-acting pupils, no nystagmus, but some difficulty in fixation of the eyes. He also complained of inability to estimate size of objects; sometimes objects and persons appeared much too large, sometimes much too small. People coming in at the door

of the ward appeared like babies. This failure in judgment of size came on usually towards the end of the day, but it could be sometimes produced by making him look steadily at a distant object.

The occurrence of hallucinations in one sensory area is favoured by hallucinations in another, thus the onset of one set might induce the other.

Auditory hallucinations were frequently combined with visual, and generally occurred in persons with an hereditary history of insanity; usually associated with delusions of persecution.

Auditory hallucinations may be simple or complex; if the latter, patients complain of noises in the ears like rushing water or a steam whistle, ringing of bells, banging and firing of guns; these may be followed by indistinct voices which become gradually more distinct; voices may be heard in the distance, or close to, or within the patient; they may relate to persons whom the patients have known and (from a real or imaginary cause) have associated with real or imaginary pains or sufferings. The voices heard are usually condemnatory and accuse the patient of immorality, vice, crime, or brutality. In females, obscene sexual expressions are frequently heard, or children crying (Cases 62, 63, 66 and 68).

An instructive example of systematised auditory hallucinations is afforded by Case 25, who said that two nurses from the infirmary, where he was first taken, continually followed him, and carried on a conversation about him, turned on electricity and pulled his legs at night. He associated the lightning pains and the cramp-like spasms with the voices that he heard talk to him. This case is of exceptional interest because for years the same delusions and hallucinations have persisted. Like most such individuals the hallucinations are worse just before going to sleep; they could be produced by covering his eyes, as I found when testing his skin sensibility. He then said he heard voices say "Too bad, Hoskyns"; again, when he heard the metronome going, he heard the voice say, following the rhythm of the metronome, "You must have been a

lunatic to run away from Sydney Street." This was precisely the same sentence which he said he heard, and of which we had a note, when he heard the metronome many months previously. This man also had delusions of poisoning, which he associated with the voices. He had had visual hallucinations, but later they ceased to trouble him, and voices were not so distinct as formerly.

Frequently auditory hallucinations occur when the individual indistinctly hears the confused sound of voices at a distance. The patients are suspicious and morbidly inclined; therefore, if they see people talking in the distance and they do not hear what is said, by a kind of morbid association the hallucinatory voices return.

Deaf people, or partially deaf people who are insane, are especially liable to hear voices and to be filled with morbid and suspicious delusions, and in the first volume of the "ARCHIVES" I recorded the case of a *deaf mute* who suffered with auditory hallucinations; this seems almost paradoxical, but he had been taught lip language and the power of communicating his ideas; therefore, it must be presumed that his kinæsthetic centres were associated with his auditory ideation centres of words, although the ordinary word perceptive centre was functionless.

Another tabo-paralytic patient, a musician, who suffered with lightning pains, heard not voices, but continually the playing of an orchestra; he associated it with the electric wires and electric currents in his body; he continually heard it whenever he concentrated his attention, and being a professional flute player, he whistled very accurately the melody he heard in his mind, and was quite surprised that I did not hear it also.

A blind tabo-paralytic woman (Case 64), who was subject to visual hallucinations, imagined that the people she heard talking, came into her bed to assault her at night, and it was these hallucinations and delusions which brought her to the asylum. The existence of organic degenerations which occasionally, even in sane people, cause rectal and clitoris crises may, in insane subjects, be a source of peripheral excitation, and be insanely interpreted.

Another interesting example of an hallucination giving rise to a systematised delusion, is that of Case 26, whose wife committed suicide eighteen months previous to his admission to the asylum. For a long time he persisted in the delusion that he had opened his wife's grave, found the coffin empty, and that she had come to him since he has been in the asylum, and he had conversed with her. These delusions with regard to dead relations are not infrequent; sometimes cases occur in which the patient has hallucinations and delusions that relatives who are still alive are dead; that they have been at the funeral and seen them buried; it is as it were a dream which has persisted as a fixed idea.

Delusions.—Delusions of persecution are common in tabo-paralytics; it is natural that they should put an insane interpretation upon their pains and sufferings, but I have been rather struck by the fact that this condition applies more to tabetic patients with associated insanity than to tabo-paralytics; for grandiose delusions, exaltation, *bien-être*, supervene very frequently in tabo-paralysis. As the mind becomes affected, and especially after an attack of mania, leaves the patients demented, they cease to complain of pains, and the cord symptoms both subjective as well as objective, are less obvious, and may even almost disappear. The subjective symptoms disappear with the progressive destruction of the seat of consciousness, and this may account for the well-known fact that paralytics do not suffer much pain from bodily injury or disease, and hardly ever complain; so that a patient may have a very severe and painful affection of the internal organs without manifesting any subjective or even objective symptoms during life. In such a painful disease as dysentery, with its attendant gripes and tenesmus, I have been struck with the fact that demented patients seldom complain or evince signs of pain.

In 28 per cent. of the sixty asylum cases of tabes, the patients were affected with delusions of persecution, poisoning, electricity, &c. In three-fourths of such patients, there were either auditory or visual hallucinations, often the two combined; and all of them, with one exception, had an

hereditary history of insanity. Nearly all the cases of hallucinations had an hereditary neuropathic or psychopathic history. The grandiose cases were apparently much less subject to hallucinations of vision and hearing, and an hereditary history of insanity was comparatively less frequent. Grandiose delusions alternate occasionally with those of persecution ; mania gives place to melancholia, and restless excitement to stupor. The grandiose delusions do not seem to be so persistent as those of persecution, and it was frequently noted in the sixty cases collected that they passed off after the patient had remained in the asylum a little while, and the acute delirium had subsided. It seemed that some were partly due to the effects of acute auto-intoxication.

The delusions of persecution associated with electricity in the body, the poisoning of food, twisting of the bowels, withdrawal of the semen (impotence), bad smells and tastes, frequently associated with hallucinations of sight and hearing, have, I conclude, an organic basis in the tabo-paralytic, and persist generally throughout the illness, although different false interpretations may be put upon the abnormal sensory stimuli. The grandiose delusions were often simply confused, incoherent, and grotesque exaggerations referring to the patient's possessions or ambitions. Thus in E. C. R. and his wife conjugal paralysis (Case 69), the prevailing delusion in both was the ownership of all the furniture of the asylum ; the man was a furniture dealer. Another man was a cutter in an army tailoring establishment ; one of his delusions was that he was a colonel in the Lancers ; another that he possessed diamonds ; but the fact that he was a Polish Jew, who had for a long time been in South Africa, makes it possible that he was once in the illicit diamond trade.

Dementia

Progressive dementia is the most constant symptom in general paralysis ; it also exists in all cases of tabo-paralysis, but as a rule, according to my experience, death takes place

before there is advanced dementia more frequently in cases of tabo-paralysis, than in ordinary cases of general paralysis. This is but to be expected, since the degenerative process which has affected the central nervous system has either been in progress in the spinal cord some considerable time, or when it attacks both the spinal cord and the brain simultaneously, death from one of the many complications arises sooner; and the fact that both cord and brain are affected indicates a more widespread process of decay. A man who has suffered with tabes for five or six years, and is then attacked with brain symptoms, as Case 46, is in a lower state of vitality than a man who starts with brain symptoms; *but if we consider that the disease is a single morbid entity, and began with the cord symptoms, then the duration is longer.* Be that as it may, it is a fact that, in cases with tabes dorsalis, at the autopsy one finds as a rule less brain wasting than in an ordinary case of general paralysis. The majority of the cases recorded exhibited either a slight or only a moderate degree of brain wasting, and we know that the dementia, generally speaking, is proportional to the extent of the brain atrophy.

The following cases, 45 and 48, were attended by left-sided seizures during life, the epileptiform fits being the immediate cause of death; these patients did not exhibit marked dementia, and at the autopsy one found very great wasting of the right hemisphere. This was particularly so in Case 45. Another case, 54, with right-sided seizures exhibited marked progressive dementia, and at the autopsy the left hemisphere was found markedly wasted, and weighing much less than the right. Seeing that the speech centres are situated in the left hemisphere in right-handed persons, it may be considered that the mental state did not really indicate a more marked dementia, but a greater difficulty in expression of ideas and the ideation of words. Although both hemispheres are simultaneously used in all mental processes, and it is not suggested that the left hemisphere is used independently of the right, even in speech, yet it is *through* the left hemisphere and its centres connected with verbal and written speech that the brain as a whole carries on all abstract processes of thought;

therefore, it must be allowed that one could ideate words and think in the abstract to some extent without the action of the right hemisphere, but without the left hemisphere this would be impossible.

The most dementia occurs when both hemispheres are wasted. In some acute cases where there has not been time for the products of decay to have been removed and absorbed, there may be extensive dementia without very great wasting, but examination of the tissue microscopically shows an acute cell destruction, generally associated with marked vascular congestion and cell-proliferation in the peri-vascular lymphatics. It was remarkable in some cases, especially Case 31, how cord and brain symptoms seemed to alternate, as if, while the process was destroying the structures in the cord, in the brain it ceased, or was in abeyance.

Note.—The morbid anatomy and pathology will be described after the cases.

CASES.

GROUP 1.

A summary of the clinical symptoms of cases of tabes which have presented some unusual clinical phenomena, or are adapted for explanation of some essential feature of the disease.

Case 1.—*Optic atrophy at* 19. *Preataxic condition for twenty-eight years, probably due to congenital syphilis, although no history was obtainable.*—(*Bethnal Green Infirmary.*)

J. E., aged 47, lost his sight between 19 and 20; he accounts for it by a blow on the head when 11 months old. Twenty-five years ago he attended St. Bartholomew's Hospital, under the care of Dr. Hensley, who then said that he had locomotor ataxy. He is very positive in saying that he had nothing to do with women before he went blind. He says that he does not see how it can be locomotor ataxy, for up to a few weeks ago he was able to walk eight or ten miles a day. Four years ago he was treated in the London Hospital for severe gastric crises. He has now gastric attacks occasionally, which last two or three days. He has a feeling of a tight cord round the waist, cutting pains in the legs, and a somewhat ataxic gait. Cutaneous sensation is nowhere lost in the legs, but light tactile sensibility is lost over the thorax and back between the fourth and tenth segments inclusive. In this region also there is some hypalgesia and considerable delay. The joint sensation is good everywhere except in the great toes. Knee-jerks and triceps jerks lost, moderate hypotonus in the legs. The pupils had had atropine put into them, they could not therefore be examined. Optic atrophy of old standing.

Case 2.—*Case of hereditary syphilis in a child, whose father died of general paralysis. At the age of* 9 *paroxysmal attacks of severe pain in the abdomen associated with vomiting and sometimes diarrhœa. A previous history of ten fits. Admitted to Charing Cross Hospital, subsequently to Darenth. Absent knee-jerks, optic atrophy, chorio-retinitis, pupils dilated, equal, inactive to light and accommodation; trunk, light tactile anæsthesia and dislocation of hip.*

P. C., aged 9, admitted to Charing Cross Hospital, May 21, 1899, for attacks of vomiting and pain in the abdomen.

Family history.—Father died at Banstead of general paralysis of the insane. It may here be remarked that the notes of his case did not refer to syphilis or any signs thereof on his body, but his offspring indicates that he suffered with this disease. Mother had two children born dead and one miscarriage. The patient began to have the attacks of pain and vomiting at the age of 7, and they became as frequent as once a week. The child also complained of pains in the bones and joints, and two years ago she had swelling of the dorsal surface of both feet.

Condition on admission.—She is well nourished, and presents signs of hereditary syphilis in well-marked rhagades on the lower lip and at the angles. Complete loss of knee jerks, but no other physical signs are mentioned in the notes, although they show that all the organs were carefully examined.

At Darenth in June, 1899, I saw her. She had then recovered from an abscess of the jaw and a pelvic abscess which had been opened; the attacks of vomiting still persisted, attended with radiating pains. Knee-jerks absent. Pupils dilated, unequal, inactive to light and accommodation.

December, 1901.—The following notes as to her condition were kindly forwarded to me by Dr. Beresford: Pupils dilated, equal, inactive to light and accommodation. Very defective vision of left eye, ophthalmoscopic examination shows chorio-retinitis and white atrophy of the disc. Optic atrophy of the right eye and chorio-retinitis. Knee-jerks absent both sides, triceps jerk present, superficial reflexes absent. Anæsthesia of chest to light tactile sensations from the third rib to the upper border of the sixth rib, extending lower in the left axilla than in the right. The rest of the body and limbs are normal to touch and pain. Very little loss of sense of position of joints. She has slight incoordination, and sways rather when standing with the eyes shut, but she has a dislocation of the hip. She still suffers with gastric crises, attended by severe pain. Her mental condition as a rule is normal, sometimes bites her tongue, and passes her urine and motions under her. At other times she has control over the sphincters, and responds to the calls of nature. She has thickening of the left femur in upper half, and a dorsal dislocation of the head of the right femur. There are no teeth in the lower jaw, and the alveolus is absorbed just like an old woman's jaw. The alveolus of the right half of the upper jaw is gone, and all the teeth, except one central incisor. Left side of the upper jaw one upper molar, first and second bicuspids, lateral and central incisor are still left. (?) Result of mercurial treatment or atrophic bone lesion.

Case 3.—Preataxy. Gastric crises of twenty years' duration still severe in character. Impotent five years.

C. E. M., sailor all his life up till the last eight years, since then a publican: later on an Admiralty messenger. Married.

Family history.—No direct neuropathic history. One of a family of seventeen children, five of whom are now alive. Patient does not know whether his mother ever had any miscarriages. His eldest brother died at the age of 50, from some brain trouble.

In 1878 he contracted syphilis (hard sore, scar still remains). In 1881 he was engaged stowing away provisions in the ship's hold, and strained himself. He had an inflammatory swelling in the groin which was lanced on two or three occasions, and from which he was not entirely free for three months. Patient not a free drinker.

Present illness.—Patient dates the onset of the present illness after the occurrence of the buboe in 1881. The first thing that occurred was a pain in the back, sharp in character, which extended both transversely and vertically all over the back: at first the pains only lasted about a day. As soon as they became acute vomiting commenced, and relieved them. There were no pains in the region of the epigastrium. At first these only came once in six months, but later became more frequent and more severe. In 1895 patient married; at this time the attacks of pain occurred about once in every three months and would last three to four days. The attacks of vomiting would last two to three hours, but now did not relieve the pains. He now began to notice that on washing his face, he had a tendency to fall down, and began to have very sharp lancinating pains shooting down his legs. After marriage his sexual powers declined. The desire for intercourse was still present, but the erection would die away without full performance of the sexual act; in fact (since 1895) he has never been able to completely perform the act. For the last three to four years he has had a sensation of wool under the soles of his feet. Also has had occasional attacks of difficulty in passing his water, not amounting to actual retention. The bowels are regular. For the last five or six years he has had a sense of constriction round his body. This feeling is synchronous with the pain. He has never suffered with diplopia. He has noticed no change in his gait nor powers of walking. His principal trouble is the increase in the frequency and severity of the pains, which have become very bad. They come on now every two or three weeks and last for a few days or weeks. Associated with the pain is intense sickness, which occurs whether there is food in the

stomach or not. This vomiting persisted on the last occasions for twenty-four and fifty hours respectively, the attacks coming on at an interval of about half an hour. It is extraordinary how soon the patient recovers and is able to take ordinary food after such severe attacks; this then, resembles sea-sickness. Patient has lost flesh considerably during the last few years, but does not feel weaker except after one of his bouts of sickness.

Present condition, January 19, 1900.—*Nervous* : (*a*) Motion—Hand grip unimpaired. Muscular power in legs good ; the muscles show no signs of atrophy, but are flabby. No incoordination of movement. Slight hypotonus of muscles in right leg. (*b*) Cutaneous sensation was charted and showed hyperæsthesia over the thorax, but no definite anæsthesia. Muscular sense unimpaired. *Reflexes*—Superficial :—Plantar reflex well marked on the left side. Plantar reflex absent on the right side. *Cremasteric*—Present on the right side, but very sluggish ; brisker on the left side, but less marked than normal. *Epigastric*—Brisk on both sides. *Deep Reflexes*—Knee-jerks absent on both sides. *Gait*—Normal. Stands pretty well on one leg. Stands erect with eyes shut. *Ocular*—Pupils slightly contracted and unequal, left smaller than right. No reaction to light, but to accommodation. Gastric crises still continue and he is now suffering from diplopia owing to paralysis of the left external rectus.

Case 4.—*Transitory paralysis of left external rectus, gastric crises, preataxic condition, knee-jerks present both sides, cutaneous thoracic anæsthesia unequal on two sides. Brother died in asylum of general paralysis.*—(*St. Pancras Infirmary, August*, 1900).

J. M., aged 45, occupation pavior and mason, work hard ; married, has had sixteen children, six of whom are alive, one miscarried, and three born dead in succession ; denies syphilis.

Family history.—Brother died in Colney Hatch two years ago of general paralysis, aged 36. He lived a very rackety life, and was the subject of syphilis, undoubted.

History of illness.—Four years ago he had double vision and paralysis of the left external rectus, gastric crises. He has no ataxy, no pains in the limbs, no paræsthesia, no difficulty of walking in the dark. The knee-jerks are present on both sides upon reinforcement, triceps and wrist tap both obtained, no loss of sense of position, joint sensation and localisation perfect. The only symptoms are double vision, slight nystagmus to the left,

Argyll-Robertson pupils and the gastric crises previously mentioned, which were extremely severe, causing him to shout out, and were only relieved by morphia. He is sick with no food in his stomach, and during these attacks he has a deep epigastric pain, and pain under the right shoulder. The vomit is very frequently bilious. There is no enlargement of the liver or gall bladder. There is some dilatation of the stomach. There is light tactile anæsthesia over the fourth, fifth, and sixth segments on the right side, and the fourth segment on the left side, which is of interest, seeing that it is on the right side that he feels the pain when the crises occur. He has no analgesia or thermal anæsthesia. His answers are reliable.

November 14, 1901.—Patient is very depressed, still suffers with gastric crises. Complains of shooting pains down the spine. He has diplopia and paralysis of the left external rectus. A. R. pupils equal 3·5 mm. He complains also of pains across forehead between the eyes (probably sympathetic associated). Knee-jerks present right side ; present on reinforcement on the left. Hypotonus more marked on the left than right. Retention of urine. Cutaneous disturbance unchanged.

Case 5.—Tabo-arthropathy, preataxic condition for twenty years.

J. L., aged 63. At the age of 43 he came to Charing Cross Hospital suffering with lightning pains, Argyll-Robertson pupils, absent knee-jerks, and a large swelling of the right shoulder joint, preceded by pain, and greatly distended with fluid. It rendered him unable to follow his occupation of stonemason. He weilds a 4-lb. hammer with the right hand to strike a chisel held in his left hand ; both the metacarpophanlangeal and the phanlangeal joints (especially of the thumbs), also the left elbow and wrists, present nodular deformities, like that of rheumatoid arthritis. The joints of the lower limbs exhibit little or no deformity. He admits having had a chancre when he was 22 years of age.

December, 1901.—The patient has been re-admitted to the hospital. He is still in the preataxic stage, knee-jerks and triceps jerks are absent, lightning pains are now present and very severe, pupils unequal, right four, left three-and-a-half mm., inactive to light, but react somewhat sluggishly to accommodation. There is no ataxy in the gait ; Romberg symptom is not present ; there is no loss of sense of position in the joints, or any marked incoordination of the lower limbs ; owing to joint deformities it is impossible to say whether there is any in the upper limb or not.

There is no trunk anæsthesia, and, with the exception of a little confusion of the prick of a pin with the touch of a finger in a few spots in the lower part of the peroneal borders of the limbs, I could discover no defect of sensation anywhere; he has never had any trouble with his bladder, or any other visceral symptom beyond the attacks of vomiting mentioned. The right shoulder joint was tapped some time ago, the swelling has therefore gone down. Skiagrams of the various joints have been made; there appears to be absorption of the head of the humerus and ankylosis. Considering the deformities of the hands there are very few osteophytes, and most of the swelling must be due to the capsules and the synovial membrane round the joints. The left elbow joint does, however, show a considerable amount of osteophytic growth. The joint disease in the upper limbs has produced a fair amount of muscular atrophy.

This case is of interest in several ways, firstly, because the diseased condition of the joints of the upper limbs was probably connected with his occupation of a mason, which he had followed for thirty years; secondly, the absence of bladder trouble and the long period during which the patient remained in the pre-ataxic stage. The sense of position in the joints, in spite of the deformities, remained unchanged.

Case 6.—*Woman. Advanced tabes, well-marked signs and history of syphilis. Brother died in asylum of general paralysis.* —(*Bethnal Green Infirmary.*)

E. C., aged 48, married. One child born dead eighteen months after marriage, no pregnancies since, papery scars all over body, undoubtedly syphilitic, occupation sewing machinist. Brother died in an asylum of general paralysis, aged 46. Her illness commenced with lightning pains, numbness in the hands, followed by ataxy.

Present state.—Marked ataxy, Romberg symptom, loss of joint sensation in the feet, legs, and hands. Deep reflexes lost, and loss of sense of position in both upper and lower limbs. Superficial reflexes, plantars absent, epigastric present. Pupils unequal, left irregular 3½ mm., right 5 mm. inactive to light and accommodation, marked hypotonus of legs.

Sensation.—Thoracic anæsthesia in area of distribution of seventh and eighth cervical upper dorsal, third, fourth, and fifth lumbar, first sacral, and hyperæsthesia in areas between. Imperfect localisation over the lower part of abdomen and

upper part of legs. Pricking with a pin is described as a touch all over the body.

Case 7.—Case of gastric crises admitted for acute intestinal obstruction. Operation contemplated; contracted pupils with inaction to light attracted attention and averted operation.

W. C. Labourer. Single. Admitted Charing Cross Hospital, August 17, 1900, for violent pains in the abdomen.

Family history.—Nothing of interest.

Personal history.—Influenza and pneumonia three years ago. Syphilis contracted fourteen years ago (hard chancre, secondary eruption, falling out of hair). Treatment (?)

Physical signs.—Has been troubled with his bladder for the last eight weeks. Has great difficulty in passing his water, and intense pain when he endeavours to micturate. Sometimes has difficulty in passing his motions. Has been a free drinker for the last two years. Patient first experienced difficulty in walking two weeks before admission. He has had peculiar sensations in the abdomen for the last four or five weeks, such as sense of constriction, like a tight cord, and at times, sensations of cramp. Shooting pains have occurred in the right leg only. Has noticed failure of sight for two years, and cannot now see to read.

Present illness.—On Friday morning, August 17, he woke with violent pain in the abdomen; this became so bad that he came to the hospital. When seen he was breathing very rapidly, and appeared to be in great distress. Pulse quite good, and he had not been sick. On examination of the abdomen, the lower part of it was found to be slightly distended, but everywhere tympanitic, except in the supra-pubic region. Abdominal movements good. On palpation nothing definite was felt. There did not appear to be any tenderness, and the abdominal wall was equally soft all over. What swelling there was, was most marked over the left inguinal region. At first there was distinct gurgling all over the belly; palpation seemed to aggravate the pain. With a diagnosis of probable acute intestinal obstruction (volvulus?) he was admitted. His rectum was then examined, but nothing abnormal was found. As the pain increased, and the patient was becoming slightly delirious, $\frac{1}{4}$ grain of morphia was given and an enema ordered. When the latter was administered, the patient was sick; after this he quieted down, breathing quite quietly and sleeping. At five a.m. he was seen in this condition, and a catheter passed without attracting his attention, and 14 ozs. of urine drawn off. Specific gravity 1·005, acid, and contained a

faint trace of albumen. At seven he vomited, and continued to do so at intervals throughout the day, the vomit being brownish acid; no blood or stercoraceous smell. The pain returned as the effects of the morphia passed off. Mr. Boyd saw patient at ten a.m., and as the pulse was good (84), he decided to wait and watch for further indications. At eight p.m. the patient was complaining of pain higher up in the abdomen, and of pain in the back and loins, as though someone was pinching him in. This, combined with the fact that the pupils did not dilate during the paroxysms of pain, led at once to a suspicion of locomotor ataxy, and on examination typical symptoms were found. While patient has been in hospital, he has on several occasions been quite delirious, and has got out of bed and walked aimlessly about the ward. It was found that these attacks were associated in some peculiar manner with a full bladder. In these delirious attacks he suffered with visual and auditory hallucinations. He stated that he heard numbers of men continually tramping up the stairs in the night, and that they came and marched through the ward like an army.

Examination of present condition. — Well-built and well-nourished man. *Motor power.*—There is no wasting of muscles, and the power in the legs and arms is good. Hypotonus of muscles is well marked. When patient is lying on his back the leg can be flexed to a right angle with his body when the knee is extended. *Sensation.*—The buccal mucus membrane on the right side, also the tongue (right half) were anæsthetic and analgesic, as well as the whole cutaneous surface of the right half of the body; but the cornea was very sensitive to the slightest touch. There is slight incoordination of the legs and arms. *Eyes.*—Argyll-Robertson pupils. There is no paralysis of the external ocular muscles. There is great diminution in the field of colour vision on both sides, and optic atrophy. Muscular sense is much impaired on the right side of the body. Gait ataxic; Romberg's phenomenon well marked. *Reflexes.*—Knee-jerks absent on both sides. Plantar, cremasteric, and epigastric are considerably diminished.

Case 8.—*Case of (probable arsenical) neuritis from beer drinking—attacks of vomiting—pains in legs and absent knee jerks.—Thought to be tabic with crises, but pupils normal.*

W. W., aged 70 years, occupation boiler maker, married, five children, one dead. He has worked in the North of England, having been sent there.

Previous history.—Has had jaundice three times, accompanied by retching and attacks of sickness. He was brought into Poplar Infirmary suffering with vomiting, sneezing, and itching of the eyes. He was unable to walk or stand, had burning pains in the legs and in the soles of his feet and cramp.

Since admission.—He has had no attack of sickness for four months, and from the first there has been a steady improvement. At the present time there is great tenderness in the calves of his legs, and the knee jerks are absent. He has had delirium tremens, and has drunk large quantities of beer. There is marked pigmentation of the legs. The *pupils react to light* and accommodation.

Case 9.—Preataxic condition. Optic atrophy, interesting syphilitic history.

G. P., aged 48. Occupation, ship's steward, hard chancre twenty-five years ago, followed by sores on the body and rash. Treated three months. Married at the time, but away from his wife on a voyage, and he remained away 3½ years. He was infected with syphilis in 1876, and returned home in 1878. His wife, who had had one child in 1873, now suffered with three successive miscarriages, one in 1878 and two in 1879. In 1880 she became pregnant, child born dead, and again in 1881, when she began to give birth to healthy children, so that she has now five healthy children living. This is best shown in the subjoined table.

First child 1873, male, alive and well.

Husband, in 1874, went for a voyage lasting three-and-a-half years ; in 1876 was infected with syphilis ; returned home in 1878.

During 1878 and 1879 the wife had three miscarriages.

Child born dead 1880.

Second child, 1881, male, alive and well.

Third ,, male, alive and well.

Fourth ,, ,, ,,

Fifth ,, ,, ,,

There is no history of nervous disease or insanity in his family ; he has never been a heavy drinker, but has suffered with malaria, yellow fever and cholera.

Present illness.—Ten years ago (that is, sixteen years after infection) he noticed he saw double for about ten or eleven days. Twelve months ago sight began to fail in the right eye, of which he has now completely lost the sight ; there is also dimness of vision in the left eye. He has complained of no shooting pains,

numbness, formication, girdle sensation, or inability to walk in the dark, but for some time he has noticed a frequent desire to make water and occasional incontinence.

Physical signs.—Optic atrophy both sides, pupils unequal, somewhat irregular, right measures 5½, left, 4¾ mms., inactive to light, active on convergence; limitation of field of vision on left side, but recognises colours. Knee-jerks right, brisk left, present; no paræsthesia, no Romberg symptom; no ataxic gait; no anæsthesia or analgesia anywhere. This case is of interest because, probably, the wife was not infected, for in 1881 she gave birth to a healthy child. It is quite possible that the three miscarriages in 1878 and 1879, and the dead child in 1880, were due to spermatic infection and defect of the male germinal plasm.

Case 10.—*Squint, optic atrophy. Loss of smell and taste of flavours; very mild gastric attacks, only very slight sensory cutaneous disturbance of thorax, preataxic condition. Knee jerk present on one side, absent on the other.*

C. F., aged 57. Occupation, barman, billiard marker, coachman, and finally shoe finisher for thirty-four years. Married at twenty, nine children in thirteen years, four alive, two died since wife died thirteen years after marriage. Ptosis and continual squint in right eye was the first symptom, occurring five years ago. Twelve months before this he had "a sore in the pipe," which healed up as the discharge was cured; the discharge only was treated. Frænum absent, two brown pigmentary patches symmetrically placed on each leg. Glands in right groin shotty. Went to Moorfields for seven weeks, and the medicine they gave cured the squint, but left the pupil large. Three years later went again owing to sight becoming misty in both eyes, and that became worse and worse (not a great smoker). Dull pain across forehead; difficulty with his work two months before Christmas, 1898. Numbness over the breast, armpits, and flanks; he knew that there was impaired sensation there. Nothing on inner side of arm. He had cramp in the calves, but no numbness in the feet. For the last few months he has experienced difficulty in passing his water; has been constipated for several years. No gastric crises. Complained of pain over pit of stomach for two years, with flatulent distension, but no sickness. The least little jerk hurt him, and he was very tender over the epigastrium. Experienced no difficulty in walking. Loss of smell since paralysis of right eye, but has a very disagreeable sensation of smell

like a drain. He can taste mustard, sugar, acid, and salt, but no flavours; therefore only olfactory affected. He cannot smell or taste peppermint or aniseed (tested). He can smell ammonia. Knee-jerk present left side, but absent right side. Hypotonus both sides, angle nearly 90 degrees with body. No loss of sense of position in legs or toes, a little loss in hands. No loss of sexual power. Used to lurch to the right when he had paralysis of the right eye. Superficial reflexes all present in trunk and lower limbs. Cutaneous sensibility, anæsthesia to light tactile impressions in the fourth and fifth thoracic segments left side, and over a region corresponding to the sacral roots. No thermo-anæsthesia either to heat or cold. Optic atrophy in both eyes, marked curving of vessels which are of normal size.

Case 11.—*Charcot's joint, both knees, in a carpet planner, who knelt all day, slight cutaneous thermo-anæsthesia, otherwise very slight symptoms of tabes.*

W. J. S., aged 56. Occupation carpet planner for twenty years (before that a carpenter) involving kneeling twelve hours a day. Married at 21, child born healthy twelve months after, alive, aged 32; twelve months later another child was born, and twelve months after that a girl, no addition since. No miscarriages. Always suffered with rheumatics, in fact for thirty years. When 18-19 had gonorrhœa which lasted three months. Patient, however, denies syphilis. Father lived to 86 years, mother to 84. No neuropathic history. Pains in legs, came on severe ten months ago. A few months later an enormous spontaneous painless swelling in the left knee: he was able to kneel on the right knee, then it became similarly affected. No pain in the legs now. No girdle sensation, difficulty with water, bowel symptoms, or gastric crises. Does not complain of unsteadiness in walking and could walk in the dark very well. Pupils, no reaction to light or pain, react to accommodation. Field of vision normal. The glands in the groin are enlarged, the right testicle is twice as large as the left, otherwise no suspicion of syphilitic residua. He has complained of pains in the legs as well as the arms, but the arms were affected later. There is not very marked alteration of sensory perception, but there is delay in appreciating light tactile sensations and slight imperfect localisation of the same, in region corresponding with the distribution of seventh and eighth cervical, upper six dorsal and the lumbo-sacral roots. There is no loss of sense of position in the hands and the patient feels quite well any

movement of the toes, and he knows exactly what is being done when they are moved. Knee-jerks cannot be tested, nor hypotonus. It was remarkable that the patient's power of appreciating the painful sensation of the prick of a pin in the legs seemed to increase as the experiments were continued, as if increased conductibility had occurred. In the limbs below the knees where painful pricking was confounded with touch sensation, one observed confusion of heat and cold; generally speaking a hot test tube was thought to be cold. When tested in the abdomen, thorax or face, he made no mistakes. There are large corns over ball of little or big toes in both feet which look as if they would soon become perforating ulcers. The Charcot knee joints are very characteristic.

Case 12.—*Tabic foot in a butler followed by progressive ataxy. Marked cutaneous sensory disturbances of the whole trunk, flaccid atony of abdominal muscles, with ballooning of the belly and drawing in of the lower ribs on inspiration through the unopposed action of the diaphragm.*

H. F., admitted to St. Pancras Infirmary 1898, aged 49, single, butler, denies syphilis, but has been in the way of getting it. When aged 38, first symptoms, ataxy and incontinence of urine. In 1897 spontaneous painless dislocation of right hip, Charcot's joint, 1½ in. shortening of thigh. Occupation, heavy place, long hours, standing, and heavy manual work.

Family history.—Nothing noteworthy, no history of previous illnesses. The following notes were made:

Ataxic gait, ataxy in arms, muscles well developed, deep reflexes both of upper and lower limbs lost, superficial plantar, epigastric, and cremasteric absent; marked hypotonus 10° beyond the vertical. Loss of joint sensation in legs and arms. *Eyes*: Two or three years before the ataxy, *i.e.*, when he was 35, he saw Mr. Critchett for double vision which got better with a course of iodide. Pupils equal 2 mm. Argyll-Robertson, sight good. He suffers with frequent attacks of lightning pains in arms and legs, and girdle sensation. There is numbness of the hands and difficulty in buttoning his clothes. No tingling on pressure of the ulnar nerve. There is disease of the right tarsus, for the foot is much swollen in that part. It is of old standing, as it came on eighteen months before ataxy.

The abdomen of this patient is enormously distended, the lateral portions hang down flaccid; the walls are like a thin mem-

brane, devoid of fat and offering no muscular resistance. When the patient takes a deep breath, the lower ribs are distinctly drawn in, but the thin abdominal walls are bulged out by the intestines which are pressed forward by the descending diaphragm.

There was almost complete absence of light tactile sensibility over the legs and trunk up to the second space, extending a short distance down the upper arm on the inner side. There was a belt of analgesia over the fifth and sixth thoracic segments, and there was also analgesia of the legs extending half way up to the knees. In the same regions there was some thermo-anæsthesia. In other portions of the trunk and extremities where there was anæsthesia, there was either hyperæsthesia or hypæsthesia. Over the forearms and hands there were many hyperæsthetic points, and in the right forearm, the seat of recent severe pains, were several greenish yellow pigmented areas with their long axes in the direction of the long axis of the limb, which he said were once bright red, and appeared when he had the pains. They no doubt correspond to the distribution of the terminal filaments of a sensory neuron, or group of neurons, which had been intensely irritated.

The very marked sensory disturbances of the whole trunk indicated a very profound degeneration of the lower dorsal and upper lumbar roots, and this gave rise to a nearly complete loss of the reflex spinal tonus. The upper cervical roots are, however, not affected; consequently, the diaphragm is entirely unopposed, with the result the lower ribs are drawn in, and the flaccid abdominal walls protrude at each inspiration. He can contract the abdominal wall voluntarily, because the path from the cortex is still open to the spinal motor cells. If we assume that the posterior roots of the lower dorsal and upper lumbar segments contain sensory fibres from the intestines, there is probably a loss of reflex tonus of the intestines, and, in consequence, paralytic distension. He suffered some time ago with rectal crises, and for some time has required medicine to open his bowels.

Case 13.—*Argyll-Robertson pupils, history of hemiplegia, knee-jerk present on paralysed side, absent on the other.*—(*Hendon Infirmary.*)

J. T., aged 69; occupation basket maker; history of a right-sided hemiplegic attack ten years ago. Married at 30, no children, several miscarriages, history of syphilis, treated for some

months with medicine. Pupils unequal, left a little larger than right, inactive to light, react to accommodation. Deep reflexes; knee-jerk obtained on the right side, not on the left; triceps ditto. Plantar reflexes and epigastric obtained on both sides, the former brisk. He has a hemiplegic, not ataxic gait. He has had no pains, and neither anæsthesia nor analgesia were discovered. He is, however, very feeble mentally.

Case 14.—*Advanced tabes, ataxy and cutaneous sensory disturbances, first symptom pronounced gastric crises, now ceased. Brother died of general paralysis.*

C. P., aged 46, married woman, husband dead, now a nurse, no children, one miscarriage twelve months after marriage, second child only lived five months, no direct signs or history of syphilis. Symptoms began with gastric crises five years ago occuring every month or six weeks, followed later by a tight feeling round the waist. Two years later inability to stand or walk in the dark; three years ago she had a hystero-epileptic fit while in St. Mary's Hospital; later she went into the Queen Square Hospital and received Fränkel treatment without benefit. Four years ago she had bladder trouble. No neurosis or nervous disease in the family, except that her brother died of general paralysis, aged 33.

Present state.—She can now get out of bed, but is unable to walk or stand without support, but she can wash and feed herself. The muscles are not wasted, there is marked hypotonus in both legs, deep reflexes of upper and lower limbs lost, superficial reflexes absent on right, just present on the left; epigastric, present on the right, absent on the left, frequently bilateral response to right-sided stimulation. Hyperæsthesia of skin over right side (*vide* fig. 8). There is a good deal of ataxy in the hands; she complains of great pains, shooting and darting in character, in the right infrascapular region. On moving, the pain was greatly aggravated, no friction was detected, although she thought she was suffering from pleurisy; pain is in the region where hyperæsthesia is indicated in the chart. Joint sensibility lost in all joints of lower limbs and in fingers of upper. She has lost sensation in the ring and little fingers of the left hand within the last three months. She first became aware of this by a cramp-like feeling. She complains of a deep-seated burning pain in the epigastrium, but *she does not now* have gastric crises. Pupils, right 5 mm., left $3\frac{1}{2}$ mm., Argyll-Robertson, complains of dimness

of vision in right eye, but no facility for examination. The sensory disturbances are indicated in the subjoined chart. In the dotted area there was blunting of light tactile sensibility and some delay; in the black part there was no response to pricking or touch, in the cross-shading there was light tactile anæsthesia, and in the one-line shading, analgesia.

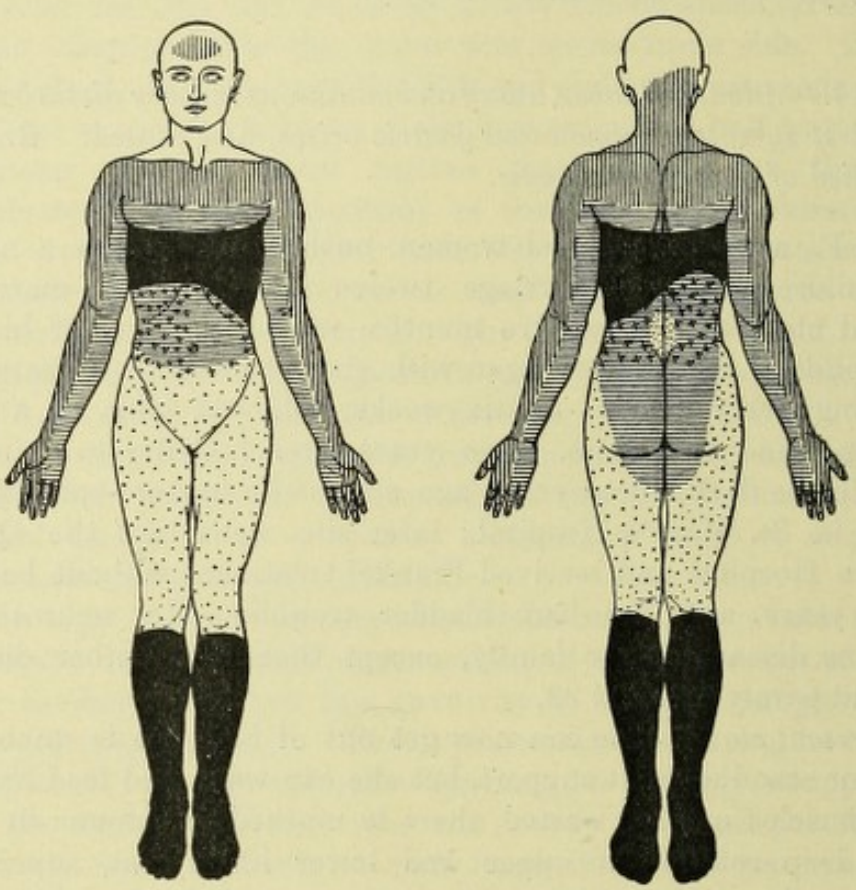
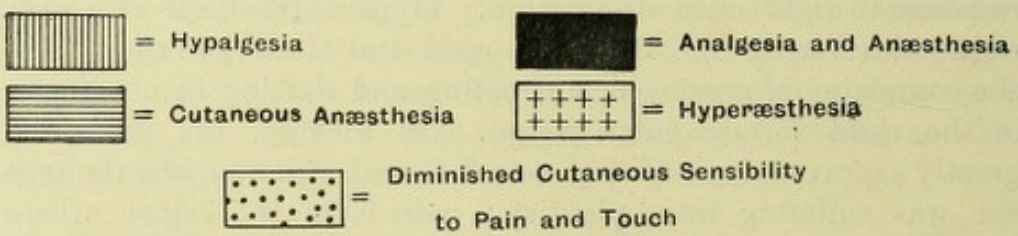


FIG. 8.



Case 15.—*Tabo-paralysis, dropping out of all the teeth, and absorption of the jaw.*

T. K., butcher, a patient in the Central London Infirmary, denied intemperance, also syphilis; but there are well-marked signs on the body, scars on the prepuce, enlarged glands in the

groin, large papery scars outside right shin. His work was that of a slaughterer and carrier. He has no friends, and his answers are unreliable as to family history. He can't remember when he had to give up his work, but attended a hospital many years ago. He has a markedly ataxic gait, Romberg symptom, absent deep reflexes of both arms and legs. Plantar reflexes absent; epigastric, right side brisk, left side present. Joint sensibility lost in toes and feet, hypotonus to a right angle. Pupils, right $1\frac{3}{4}$ mm., left 2 mm., Argyll-Robertson. He tells me all his teeth fell out not long ago, and there is great absorption of the alveolus in both upper and lower jaws. His mental state is such that his sensibility could not be tested with accuracy. He is unable to do the simplest calculation, his memory is very bad, mental reaction very slow, orientation in time and place very defective. He has a childish, fatuous expression, but his speech does not seem to be affected. There is no tremor of tongue or face. His mental condition suggests progressive dementia.

Case 16.—*Preataxic condition, brisk knee-jerks, gastric crises, gradual development (with each successive crisis) of belt of thoracic anæsthesia.*

F. B., aged 37; packer in tobacco. No neuropathic family history. Definite history of syphilis sixteen years ago with secondary symptoms, for which he was treated eighteen months; came to Charing Cross Hospital with paralysis of the right external rectus, Argyll-Robertson pupils, pains in the legs, first felt three years ago, history of several gastric crises, knee-jerks present and exaggerated, no Romberg symptom, no ataxy, no girdle sensation or difficulty with the bladder. Diagnosis, preataxic tabes. Eighteen months later, July, 1901, was seen again, suffering with pains in the legs. There was no change in his condition noticed. There is no squint now, the knee-jerks are still brisk, there is no trace of ataxy and no hypotonus. A little later, I found a patch of anæsthesia corresponding to the posterior branches of the fourth and fifth dorsal roots on the right side. The anæsthesia came on after a series of gastric crises, with local pains. Three weeks later, he again suffered with a severe attack of gastric crises, and on testing the cutaneous sensibility, I now found a complete belt of cutaneous anæsthesia corresponding to the fourth and fifth segments as shown in the accompanying chart (fig. 9).

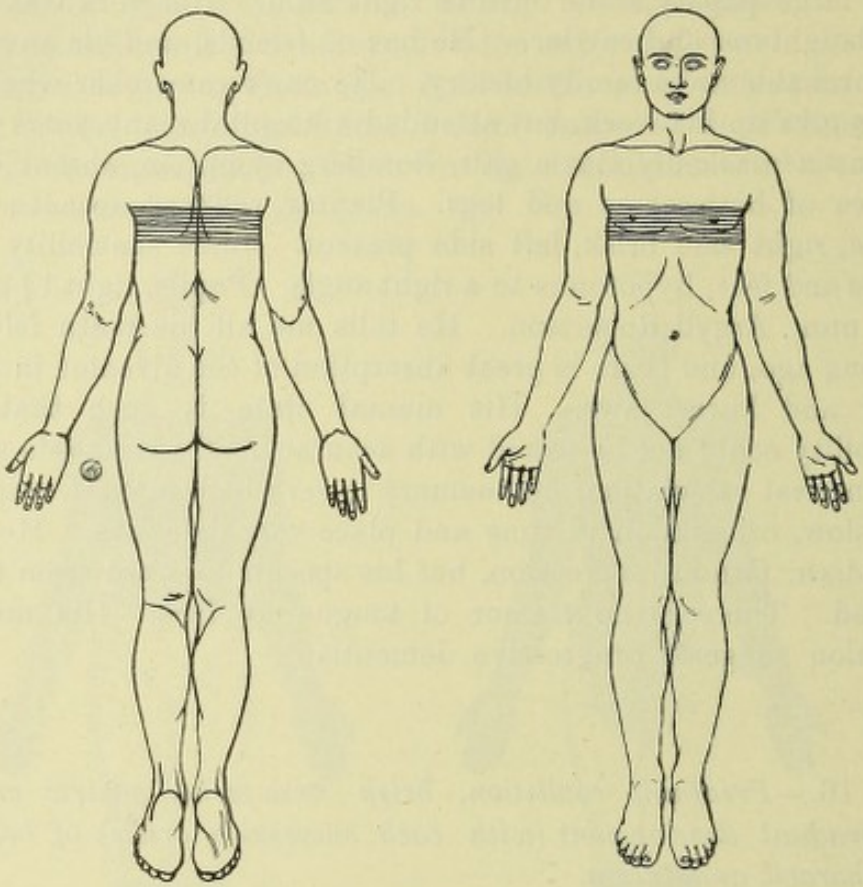


FIG. 9.

= Cutaneous Anæsthesia

Case 17.—*Tabo arthropathy. Gastric crises. Husband died in an asylum, of an acute brain disease, at the age of* 36, *very probably general paralysis.*

B. C., aged 53, an inmate of Lewisham Infirmary, married at 17, no children born alive by the first husband; four months after marriage, miscarriage; (2) miscarriage six to seven months; (3) miscarriage four months; she has always been in good health, she gives no sign or history of syphilis, except the history of miscarriages, and the existence of a *pigmented scar*, size of a half-crown, over the upper part of left buttock. Her husband died five years after marriage, at the age of 36, in an asylum, of acute brain paralysis. She remained a widow four years; married again, and had no children by her second husband.

Occupation.—After second husband died, she worked a heavy sewing machine for four years. She struck her knee against the wheel, and pain in the knee followed, but she had to go on work-

ing the machine to earn her living. The knee swelled up, but caused her no pain, she went to the hospital, and the surgeon informed her that it would have to be amputated, which surprised her very much. A year later, it was taken off at St. Thomas's owing to the foot beginning to swell. Mr. Shattuck has informed me that it was a Charcot joint. There were no signs of ataxy then. After leaving the hospital, she worked a hand machine for three

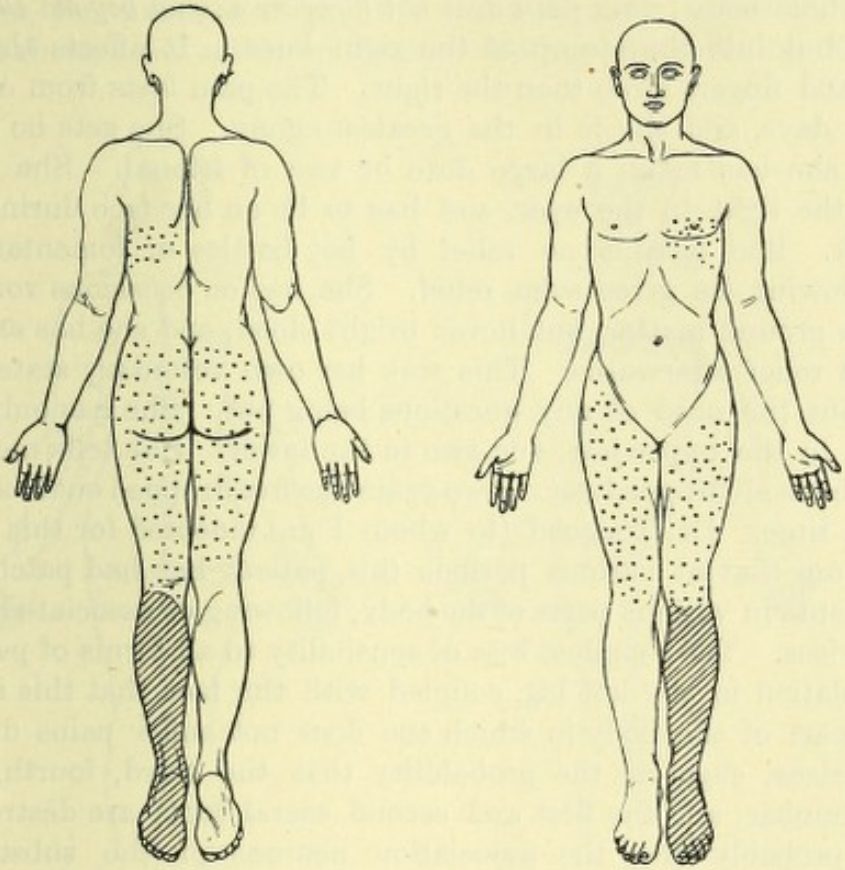


FIG. 10.

or four months, but she was obliged to give it up owing to attacks of sickness. These attacks came on independently of food. At the time she had her leg off, she had the Argyll-Robertson pupils, but no affection of the bladder or bowels.

Physical condition.—Pupils unequal A.R. right 3 mm.; left 5 mm. Colour vision good. No contraction of fields. Reads small print. No affection of other *special* senses. *Musculo cutaneous.*

—Feeling of wool on the sole of the foot. Pins and needles of left hand. Marked loss of sense of position in joints of lower limb; not marked in hands. No great amount of muscular wasting, but *marked* hypotonus of left leg. Knee-jerks absent. For sensory disturbances of skin, *vide* charts. *Visceral symptoms* —She is subject to very severe gastric crises, which are preceded by intense occipital headache, and a feeling of distension of the stomach, and this is followed by attacks of lightning pain through the whole body; *this pain does not however extend beyond the left knee*, but into the stump of the right knee. It affects the left arm and fingers more than the right. The pain lasts from one to three days, and she is in the greatest agony. She gets no relief until she has taken a large dose or two of trional. She can't bear the light to the eyes, and has to lie on her face during the attack. She obtains no relief by hot bottles or fomentations. Swallowing ice gives some relief. She has on occasions vomited coffee ground matter, but never bright blood, and she has experienced relief afterwards. This was her own voluntary statement without the need of any questions being put. She has only one tooth in the upper jaw, and two in the lower. She tells me that they have all dropped out. Two years ago five dropped out in a very short time. Dr. Toogood (to whom I am indebted for this case) tells me that at various periods this patient has had patches of erythema in various parts of the body, following or associated with the crises. The complete loss of sensibility to all forms of painful stimulation in the left leg, coupled with the fact that this is the only part of the body in which she does not suffer pains during the crises, suggests the probability that the third, fourth, and fifth lumbar, and the first and second sacral roots are destroyed, and probably also the association neurons of the substantia gelatinosa in these segments.

Case 18.—*Female. Definite syphilitic history, tabo-arthropathy, other symptoms of tabes slight. Cutaneous anæsthesia of fourth segment, but posterior divisions unaffected.*

A. C., aged 57, married at 24, no children living, four pregnancies: (1) miscarriage six months after marriage; (2) born dead twelve months after marriage; (3) born dead; (4) miscarriage. Says that she had good health all the time, but hair came out soon after marriage; she suffered with sore throat and (rheumatic?) fever.

History of illness.—First symptom was a swelling of the left knee twelve years ago, came on gradually, very painful, she attributed it to kneeling as a charwoman. She went on using it to kneel on for eight years. For the last ten years she has suffered with pains in the legs. She had no ataxy. Two and a half years ago her left leg was amputated for Charcot's joint. A little later the right knee began to swell; it was very painful, and she had to come into the infirmary again. She complains now of pains in the hand, and she has marked ulnar deviation and typical rheumatoid arthritic affection of the phalangeal and meta-

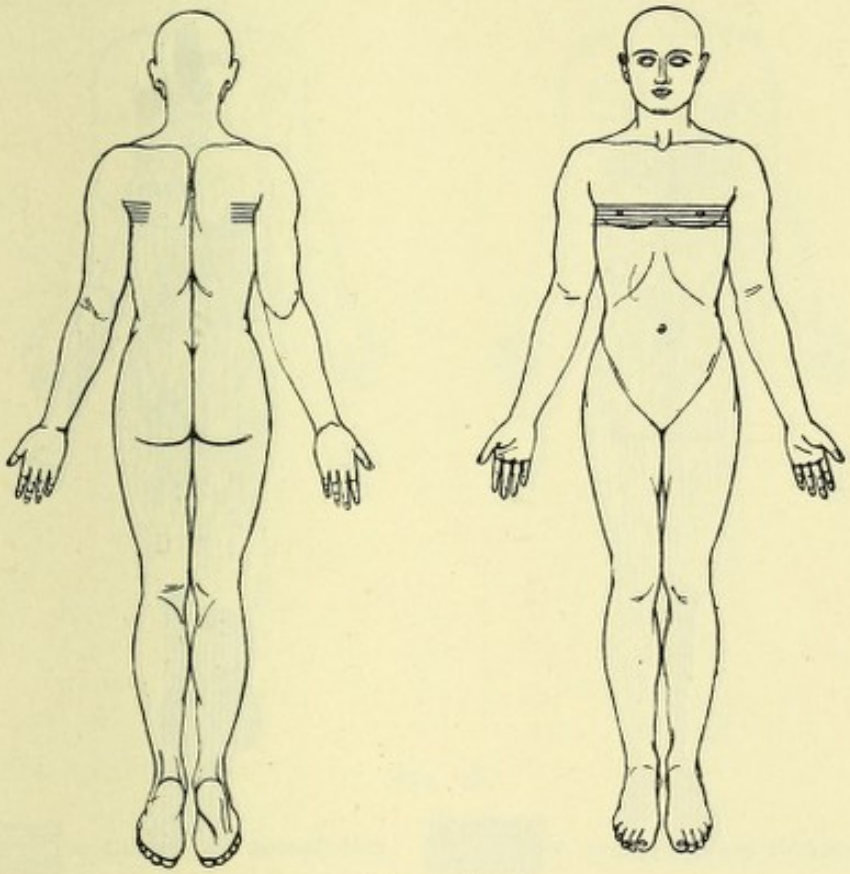


FIG. 11.

= Cutaneous Anæsthesia

carpophalangeal joints. The deep reflexes, triceps and knee-jerks are absent. All the superficial reflexes are present. There are many papery scars over the back and legs; the right knee-joint is much enlarged, there is considerable fluid in the joint, and it is painful. She suffers with no ataxy of hands or feet, or loss of joint sensation. Cutaneous sensibility (see chart). Note pos-

terior division of fourth thoracic root not affected. Vision good, colour vision good. Three years ago she suffered with transitory diplopia. Pupils equal, 4 mm., A.R.

Case 19.—*Ataxy, impotence with anæsthesia of genital organs and anal region, pointing to destruction of sacral and coccygeal roots.*

R. D., aged 38, carpet planner, admitted for unsteadiness in walking, shooting pains in legs, and tightness round the waist.

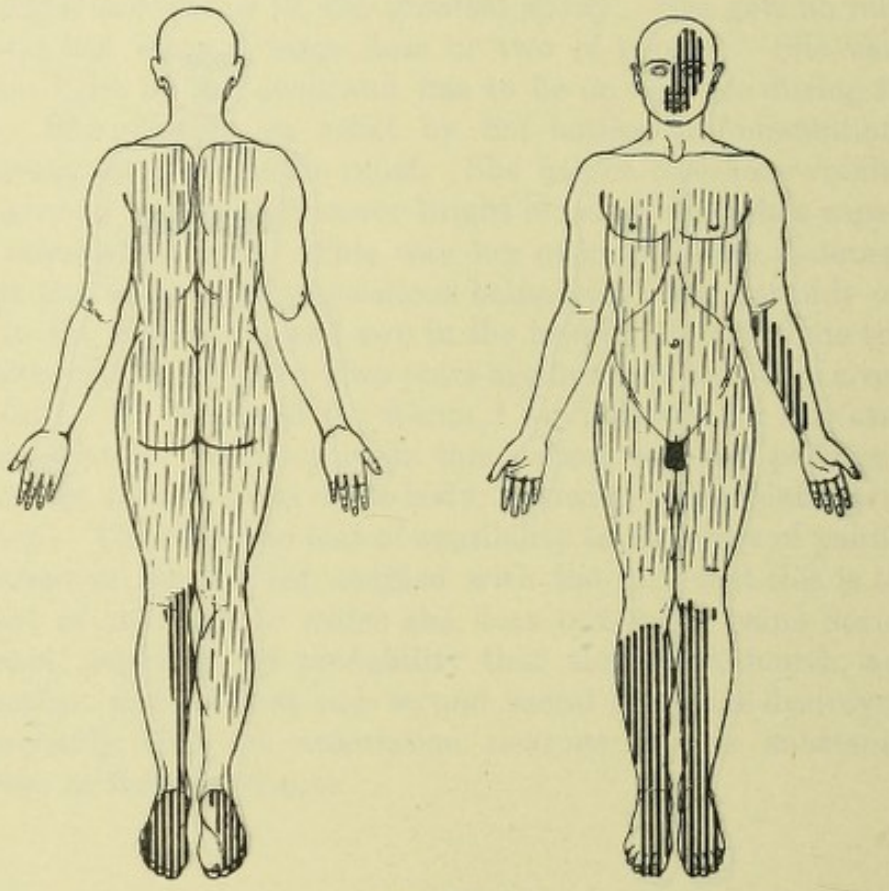


FIG. 12.

Family history.—*nil* noteworthy, no neuropathic heredity.

Personal history.—Married fifteen years. His wife has had nine children, six of whom are living.

Previous illnesses.—Syphilis two years before marriage, seventeen years ago suffered with hard chancre for which he was treated for two months. Patient has led a hard life, he has

been a carpet planner since he was twenty years of age. Moderate alcohol.

Present illness.—Began about a fortnight after recovery from influenza, January, 1900, by shooting pains in both legs and pain in the head of a morning. Between the attacks of pain he noticed numbness of the legs, and felt as if he were walking upon felt. He commenced to be unsteady in his gait, and frequently

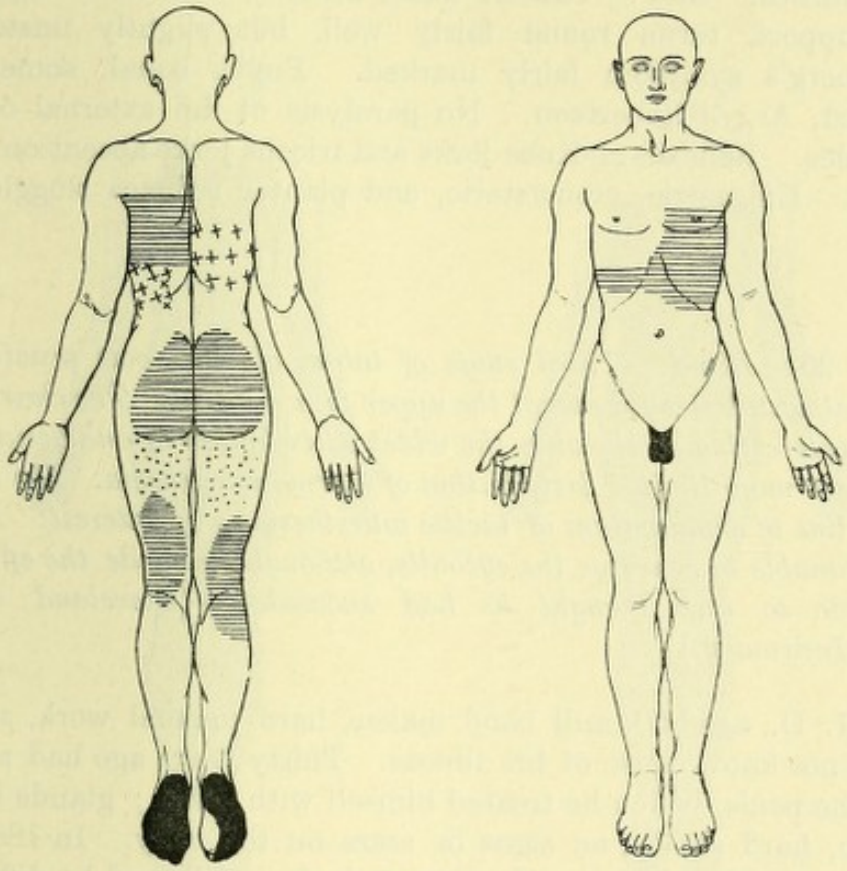


FIG. 13.

pitched forward as if his knees had given way. About this time he had very little power in retaining his urine, which would dribble away unless he frequently relieved his bladder. One month after the onset of the first symptoms, patient felt a numbness in the anal region, and he became very constipated; had also pain in the perinæum, especially when he micturated.

Feeling of constriction round the lower part of the abdomen for the last six years, not worse lately. Complete failure of sexual power for the last six months. No gastric, laryngeal, or nephritic crises. Muscular power unimpaired, some hypotonus of hamstring muscles. Sensation :—Considerable impairment of tactile and painful sensations in regions indicated in chart, but thermal sensibility unimpaired (figs. 12 and 13). Muscular sense unimpaired. Very slight incoordination of upper and lower extremities. Gait :—Patient walks steadily without a wide base of support, turns round fairly well, but slightly unsteady. Romberg's symptom fairly marked. Pupils equal, somewhat dilated, Argyll-Robertson. No paralysis of the external ocular muscles. Reflexes:—Knee-jerks and triceps jerks absent on both sides. Epigastric, cremasteric, and plantar reflexes sluggish.

Case 20.—*Blind. Third stage of tabes, all the roots practically almost destroyed except the upper four cervical. The charts are interesting as showing the wider distribution of analgesia, and the more limited distribution of thermo-anæsthesia. The sharp line of demarcation of tactile anæsthesia is of interest. He is unable to converge the eyeballs, although he made the effort to do so and thought he had succeeded.*—(*Cleveland Street Infirmary.*)

H. D., aged 50, mill band maker, hard manual work, single, does not know cause of his illness. Thirty years ago had a sore on the penis, which he treated himself with lotion; glands in the groin, hard shotty, no signs or scars on the body. In 1882, he suffered with cold feet and pains in the legs, followed by difficulty in walking, double vision, and inability to distinguish colours. He had to give up work in 1883, because he was too unsteady on his legs, sight progressively became worse, and six or seven years ago he became quite blind. About this time he also had difficulty with his water. He is now quite unable to stand, walk, or feed himself, partly because of the loss of sense of position and partly through muscular wasting and weakness. He is *much emaciated*, the feet are very cold and in a position of talipes equino varus; they are cyanosed, and the skin smooth and glossy. He is unable to turn himself over in bed, and is quite helpless. All the deep and superficial reflexes are lost in the limbs, and there is marked hypotonus. He has no joint sensations, and he cannot tell

whether there is anything in his hands or not when it is put there, and the only way he knows is to put the object up to his lips and feel with this sensitive part of his body. Thus I put the percussion hammer in his hand, made him clench his fingers (he held it between the ring and middle fingers with the fist closed), but not till he had put his lips to it could he tell me that it had a stem. He is absolutely blind, right pupil 6 mm., left

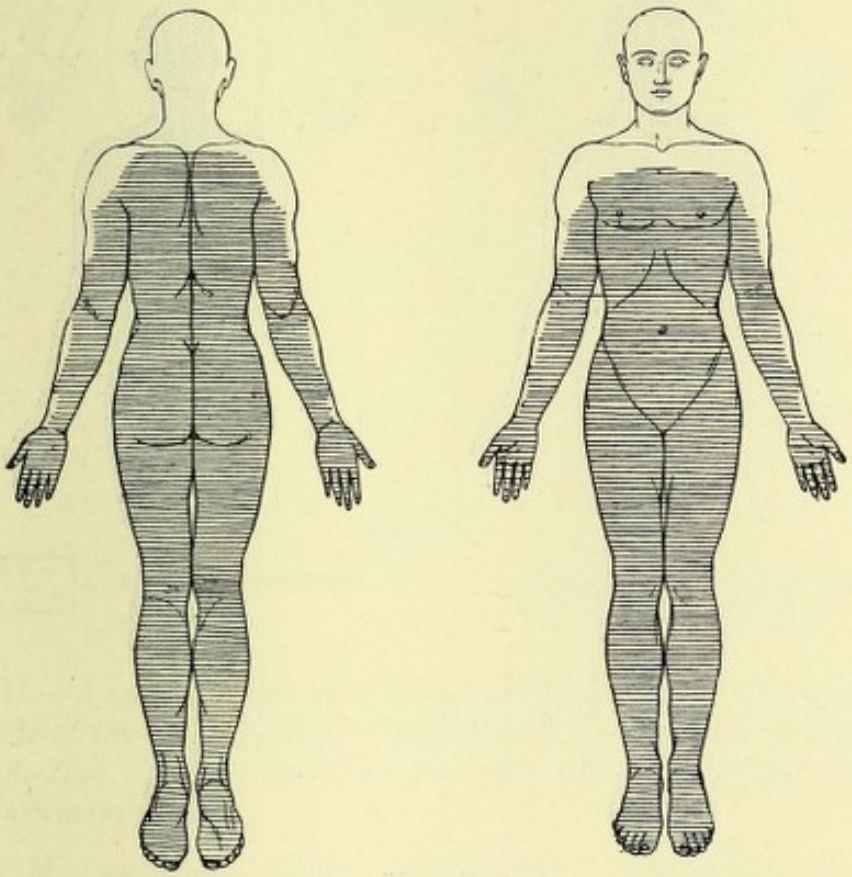


FIG. 14.

= Cutaneous Anæsthesia

5 mm., cataract right eye, both pupils inactive to light and pain, and hardly any movement on convergence. On being told to lift up his left hand and look towards it, the right eye remains immobile. The left moved towards it, and then immediately swung back by oscillations to the straightforward position. The patient thought that he was directing his eyes to the hand all the

time he was making the effort to do so. He was unable to converge the eyes to the position required for looking at the nose, although he made the effort, and he was rather surprised when I told him that he had not moved his eyes. He has no affection of taste or smell; the cutaneous sensibility is indicated in the accompanying charts. It may be mentioned that there is always

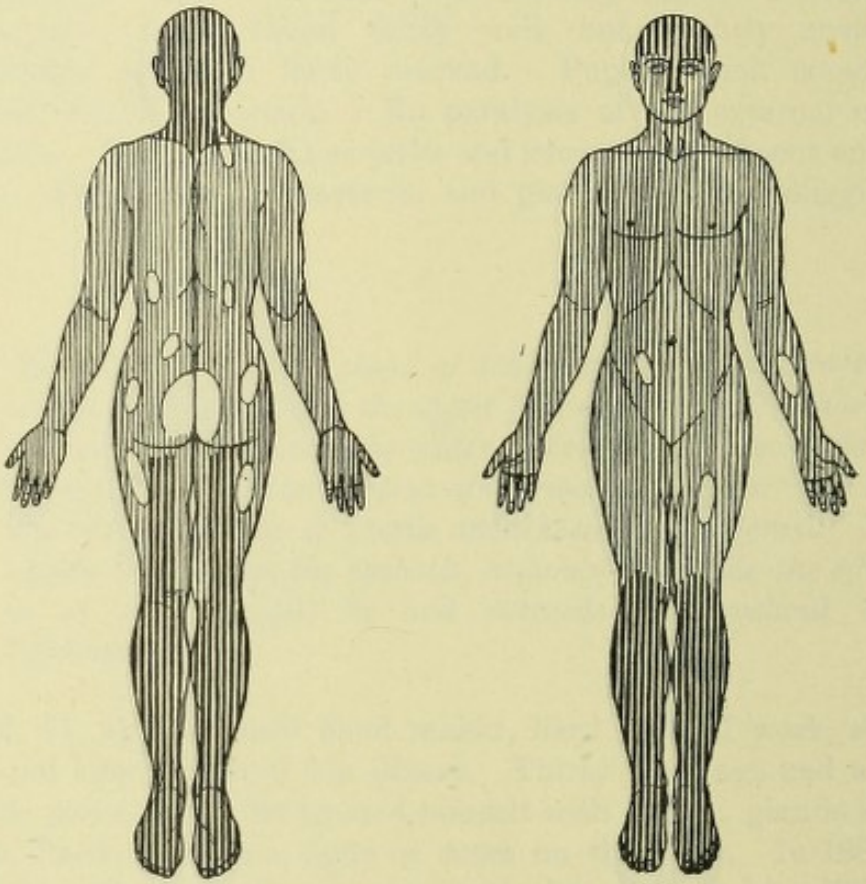


FIG. 15.

considerable delay where sensation is felt at all, generally incorrect localisation, sometimes allochiria. There is a good deal of variation with regard to response to stimuli, as if the stimulus was sometimes insufficient to rise into consciousness, the stimulus sometimes became apparent by repetition and summation. Places where formerly he felt prick of pin were afterwards not felt, while other places were.

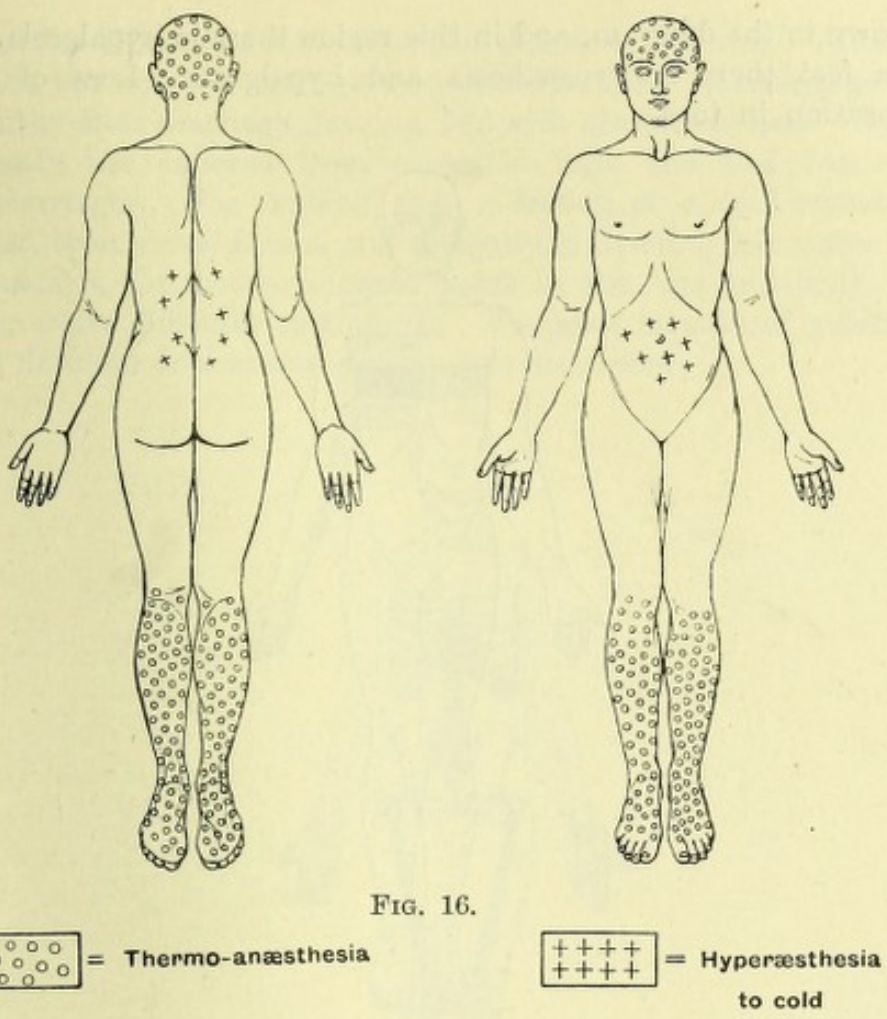


FIG. 16.

Case 21.—*A case of tabes to show extension of anæsthesia over one side of thorax and down inner side of left arm, showing unequal affection of the roots on the two sides.*—(*Bethnal Green Infirmary*).

E. M., aged 34, widow, charwoman. Married at 19, no children, never had any, history of three miscarriages, first being four or five months after marriage, marks of old syphilitic eruption on the body, and a squamous syphilide. Symptoms commenced two years ago with a giving way of legs, lightning pains and bladder trouble. She can walk fairly well, knee-jerks are absent even on reinforcement, there is considerable hypotonus in the thigh muscles, deep reflexes lost, superficial present. Pupils, irregular, slightly unequal, about 5 mm., very sluggish reaction to light, react well to pain and accommodation.

Cutaneous sensation.—There is anæsthesia and hypæsthesia over the left side of the thorax and extending down the inner side of the arm. There is a belt of cutaneous anæsthesia, as

shown in the diagram, and in this region there is hypalgesia. In the feet there is hypæsthesia and hypalgesia. Loss of joint sensation in toes.

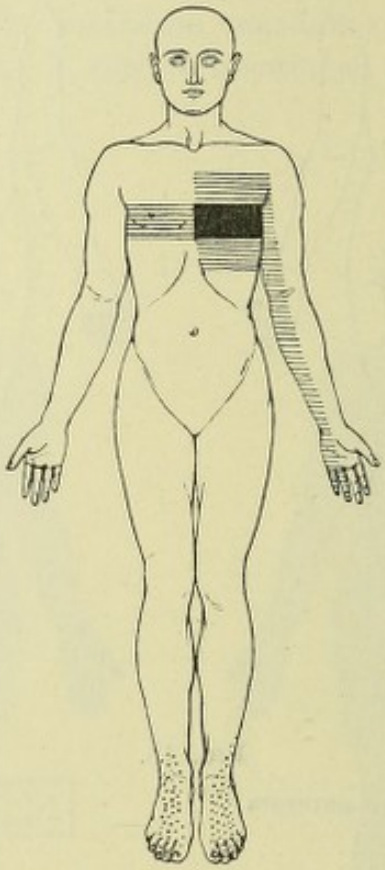


FIG. 17.

= Analgesia and Anæsthesia

= Cutaneous Anæsthesia

= Diminished Cutaneous Sensibility to Pain and Touch

Case 22.—Female. History of syphilis from husband at 18; married second husband four years later, miscarriage, ulcerated legs, rectal crises, bladder troubles, and symptoms of mild gastric crises, loss of use of legs, symptoms of progressive tabes, marked cutaneous disturbances of trunk and lower limbs progressing rapidly with severe pains, loss of bone sensibility in same region as cutaneous disturbances. Mental depression, attempted suicide.

C. A., aged 37, married woman, went to the Obstetric Department of Charing Cross Hospital, and was sent on to me.

Personal history.—Married at 17 to a retired naval man; eight months' child born, lived only two days. She found that her husband suffered with venereal disease, she had a sore

throat and her hair came out; she therefore left him. After his death, she married again at 22 a retired soldier. Miscarriage four months after marriage, leaving her with uterine disease. Subsequently she suffered from ulcerated legs, and had two more miscarriages. She noticed first a feeling of a cord round the waist, then rectal crises, and difficulty in holding her water two years ago, for twelve months pains in the legs and body, and progressive difficulty in walking. Frequent attacks of giddiness and flatulent eructations with gastric distension.

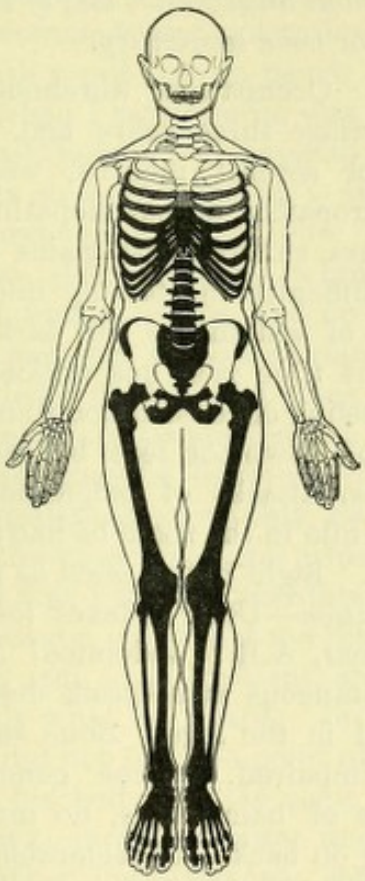


FIG. 18.

Black indicates the bones which are insensitive to the vibration of the tuning fork. The third and fourth ribs were not absolutely insensitive. In distribution the loss of bone sensibility in this case corresponded pretty closely with the cutaneous anæsthesia.

Physical signs.—Pupils equal, small, A.R.; absence of deep reflexes, plantar reflexes lost, epigastric just present, cutaneous anæsthesia from fourth to tenth segments inclusive, anæsthesia and paresthesia of soles of the feet, analgesia or hypalgesia to pricking of legs, and of the thorax from fifth to ninth segments, with some hyperæsthesia above and below. Marked incoordination and wasting of legs, no incoordination in hands. Marked hypotonus of hamstring muscles.

Two years later she became quite helpless, the cutaneous disturbances had become more marked. Slight contraction of field of vision, and pallor of discs. Retching attacks without vomiting, epigastric pain. Foot drop from paralysis of dorsal flexors, some inversion of soles, especially of right foot, from peroneal paresis. Loss of bone sensibility, as shown in the above chart. She is now very depressed because she fears she may give her husband the disease. She has attempted suicide.

Case 23.—*Ataxy without anæsthesia, loss of sense of position, without loss of joint or bone sensibility.*

H. M., aged 39. Occupation, warehouseman. Married, no children, one miscarriage three years ago. Contracted syphilis in 1886, treated for several months, well-marked secondary symptoms. No neuropathic or psychopathic history in family. For four or five years, suffered with pains in the legs. During past year has had difficulty in starting micturition, bladder not emptied. Difficulty of walking in the dark and sways with his eyes shut. For some time past has noticed that his legs would suddenly give way, feeling of constriction round the waist, shooting pains up the spine and down the legs, but never extending to the head. Gastric crises, attacks of palpitation, rectal crises, and laryngeal crises. While in the room he had an attack of laryngeal spasm and dyspnœa. Sight not so good as it was.

Physical examination—Deep reflexes lost. Ataxic gait first stage. Pupils unequal, A.R.; abdominal and plantar reflexes exaggerated. No cutaneous anæsthesia discovered, hyperalgesia over the thorax and in the legs. Bone sensibility unimpaired, joint sensibility unimpaired. Some general loss of weight. Hypotonus moderate of hamstrings, no muscular wasting. No foot-drop when lying on back. Considerable static ataxy in legs. Commencing grey atrophy of both discs.

GROUP 2.—Cases of Tabes with Insanity.

Case 24.—*Tabes* (*? tabo-paralysis*), *with grandiose delusions, mania à potu, auditory hallucinations, analgesia. Tabic foot.*

J. B., aged 38, admitted to Hanwell, April 2, 1900, occupation musician, single, duration of attack two weeks. He was admitted to the asylum in a very excited, restless, talkative, and threatening condition. He states that he is the greatest composer and flute player in the world. He fancies he has £80 in his pocket, which is not true, also that he possesses great wealth, horses, carriages,

&c. He talks incoherently of solicitors, detectives and numbers of police at his command. He has been employed in the orchestra of a theatre and he has led a very irregular dissolute life. Diagnosis was *mania à potu* and probably tabo-paralysis.

Personal history.—He has twice before suffered from melancholia, the first attack occurring seventeen years ago.

Family history.—Mother died of phthisis, and father of paralysis, aged 61. Family history of intemperance. Patient's relatives very neurotic individuals (Dr. Alexander).

Medical examination.—Tongue coated, very unsteady, pupils equal and react to accommodation, not to light, knee-jerks absent, history of syphilis with small scar on penis.

In August, 1900 when I saw him he was in the following condition :—Stepping gait, but can manage to get upstairs, absent knee-jerks, hypotonus in legs, Romberg's symptom. He has an enlargement of the tarsus on the right side. He is much quieter, mentally, than he was, sleeps well and is quiet and well-behaved. There is no tremor of tongue or lips, and no affection of speech is noticeable. Patient can give a good account of himself and his family, and his knowledge of time and place are alike good. The only evidence of insanity is a certain amount of loss of control and the repeated statement that he hears electric wires playing continuously. When questioned about this, he whistled an air from an opera to indicate the tune which the wires were playing. He was much astonished that I did not hear this music, but his listening attitude and expression convinced me that these *auditory hallucinations* were very real to the patient, and that what he was whistling was actually what he heard. In testing his sensation it was found that he did not feel the point of a needle except as a touch in any part of his body. After some delay he recognised a hot spoon. He has no girdle sensation and no visceral crises.

October 24. Further improvement, sent out on trial. He was not heard of again.

Case 25.—*Tabes dorsalis, with ataxy and left crural monoparesis, auditory hallucinations, systematised delusions of persecution associated with the pains ; little or no dementia.*

F. W. R., male, aged 35, single, clerk ; admitted to Claybury, September, 1899.

Family history.—Mother died in asylum, father attempted suicide, died of gangrene.

Personal history.—Gonorrhœa several times, syphilis at the age of 26, urethral chancre, followed by syphilitic eruption treated for

five months. Two years ago right great toe removed for diseased bone. He began to feel electricity in his legs about two years ago.

Mental state.—"Female enemies follow him about; they work the electricity on him; the whole thing is going on owing to a jealous woman, who has followed him from Plymouth to Homerton, thence to Claybury." He hears female voices talking every night; they talk to one another, and he gets quite confused and helpless; they are the voices of two nurses from Plymouth; he is watchful, and "has to keep his eyes open"; his memory and orientation are good, and he can talk on most matters quite sensibly.

Physical state.—On both shins numerous old circular punched out scars of former syphilitic ulcers; cervical glands shotty; pupils equal, 2½ mm., react slightly to accommodation, doubtfully to light. Knee-jerks absent; sensation is dulled and delayed in the feet and legs, but localisation fairly normal. Right mid-thigh measured 12 ins. in circumference, left, 11¾ ins. Right mid-calf is 8½ ins. in circumference, the left, 7¾ ins. There is no loss of sense of position or cutaneous sensibility in the arms, but reaction time is lengthened. Plantar and abdominal reflexes present. He can hardly stand with his eyes open, and would fall when they are shut; his gait is tabetic; he stamps with the right foot when walking, and drags the left foot after him; there is hypotonus in both legs; there is some fine tremor of the tongue, but no jerking; there is no tremor of the face or lips, and no affection of speech. Later he developed a slight external squint in the right eye.

I saw this patient August 30, 1901. He walks with a wide base, heels down first, throws the right leg forward like a hemiplegic man, dragging the paretic left. He is unable to stand or walk without assistance; all superficial reflexes present; triceps and knee-jerk absent; sense of position lost in toes and ankle of right foot, not in left. Pupils, 3 mm., trace of light reaction in the right eye, none in the left; accommodation in both eyes. Cutaneous hypæsthesia in the area of the fifth thoracic segment; hypæsthesia and hypalgesia of the right foot, none in the left.

Mental state.—He answers questions quite rationally, but tells me that he hears voices at night talking to him; they are those of women, one particularly, a nurse, who continually annoys him, not only by what she says about him, but by causing him to have electric shocks in his limbs, body, and face. They pull his bowels about, and cause him to have pains at his heart; some time ago they continually put poison into his rice pudding, which burnt the

inside of his stomach. He has nothing to complain about his food now; his senses of smell and taste are both good (tested), also his hearing in both ears (tested).

April, 1902.—Physical condition shows little change, except that the general health is better. He is still very ataxic, and walks with a hemiplegic gait; his sensibility to light tactile sensations on the chest showed at first anæsthesia, but after repeated observations it became only a condition of hypæsthesia of the sixth, fifth, fourth, third, and second dorsal root areas, the anæsthesia extending down inner side of the upper limbs as far as the middle of the forearm. The better appreciation of the cutaneous stimulus was no doubt an effect of summation. While performing this test his eyes were covered, and he was requested to put his finger on the spot touched. A metronome was beating at the time, and he said that he heard voices say to him, "You have been a lunatic to have gone away from Sydney Street." Last year he made the same statement when the metronome was set going, and he said it in rhythm. He hears the female voices especially at night, but also in the airing court, where they appear distant, but very real; when I spoke of them as hallucinations he was much disturbed. He has the same delusions that the people whose voices he hears torture him with electricity, and unasked he said "that they had now turned the electricity on to his arms," affecting his ring and little finger, which is of interest, seeing that the cutaneous anæsthesia has now spread to the arms, indicating thereby degeneration of the lowest cervical and uppermost dorsal roots. He says that "they pull his legs at night," which is, no doubt, an insane interpretation of spasmodic cramps; there is loss of joint sensation in the ankle and toes, specially in the right, by a failure of the synergic action of the dorsal flexors of the foot when the hip is flexed; considerable hypotonus in the legs; no affection of speech.

Case 26.—*Early tabo-paralysis, (probably) drink. Delusion of wealth. Hallucinations and delusions respecting his wife who committed suicide.*

S. J. P., aged 36, admitted to Hanwell, August 8, 1901. Occupation, coachman; married, no children.

Family history.—Father and mother died when young, no history of insanity or consumption obtainable. Brother and sister alive and well.

Personal history.—Since wife committed suicide eighteen

months ago, he has drunk a good deal. Up to then he had been quite healthy and temperate. About three months ago he suddenly developed ideas of wealth, and made all manner of statements with regard to the great deeds he had performed. He has always been good-tempered but excitable. History of chancre, treated at Lock Hospital four years before marriage. He has many delusions of wealth and grandeur; also that his wife has come to life again, persists that he saw her last Saturday week, and that she has been seduced by numbers of people. He knows where he is, the time of the year, and beyond the delusions mentioned, gives an accurate account of his previous life. Speech is unaffected, and there is no tremor in the lips or tongue. There is no ataxy, the right knee-jerk is absent, the left just obtainable. Pupils irregular, 3½ mm., Argyll-Robertson. No cutaneous anæsthesia.

He made the following statement to me :—

His wife came both to St. Pancras Infirmary and to Hanwell to see him, she was fairly well dressed, and looking well. She is now an actress at New York, but had never been an actress in England. She was buried from her own house, and he worried about her, and said he would have the coffin opened, and when they got there, they found the coffin empty. When she came to see him she had nothing on except a shirt, and a Mr. F. gave her clothes and took her straight out to New York. He has written a letter, but it has been returned. I asked him, "Did she talk to you when she visited you." He replied: "Yes. She said: 'We shall not have the old happy days over again,'" he said, "It was certainly not a dream."

April 1902.—Physical condition good, but he still persists that his wife is alive, and visits him occasionally, and that he knows she is not dead, for he had the coffin opened, and it was empty. He will talk quite rationally on other matters.

October 28.—Slight tremor of face and tongue, expression of early general paralysis. Slight dementia. He has now lost delusion about his wife.

Case 27.—*Tabes, optic atrophy, ataxy first stage, formication and sensation of water trickling in his skin, which he believes is the medicine he had at the London Hospital. Trunk anæsthesia and hypalgesia. Later crises either larnygeal or cardiac.*

J. D., married, aged 24. Denies ever having syphilis, but has been in the way of getting it, admits that he had a veneral sore, not treated. His wife has had four children born alive, of

which two are dead; one born dead, and three miscarriages. There is an inherited neuropathic history on the father's side. Patient himself has always been an anxious man; good husband, and good father, not addicted to alcoholic excess. He has complained for the last five years of rheumatic pains in his legs, and two months ago was unable to use his right arm on waking in the morning. He noticed that vision in his right eye was greatly impaired. For some time past his wife states, that he used to come home after his work greatly fatigued, and wanted to sleep. She never noticed any strangeness in his manner. He was always quite sane and rational, although very depressed. His wife noticed that he has had a staggering gait for more than six months past. Three weeks ago, he went to the London Hospital on account of his affection of sight. The curious sensory phenomena that he now complains of, he had then, and at first it was thought, specially as he had concentric limitation of the field of vision on the right side, that his disease was of hysterical nature. The Argyll-Robertson pupils, the gray atrophy, the absent knee-jerks and his mental depression led Dr. Head to consider the case to be either one of tabes with mental crises, or early tabetic general paralysis. He was sent to the Whitechapel Infirmary, and transferred to Claybury. He describes what took place when he appeared before the magistrates and was certified, and persists in the statement that he feels the sensations he described, that they are no delusions, and that owing to his having stated these sensations as being real, and due to the medicine he had given to him in the London Hospital, the magistrate was satisfied as to his insanity. He relates that another patient in the infirmary ward, told him that even if he felt those sensations he should not have mentioned them, but "he had to speak the truth." This morning, September 25, he said that he had the feeling of water trickling down his right leg, and a sensation as if his foot were swollen. He may have had this sensation; when, however, shown the right foot, and asked to compare the size and appearance with the left, in which he had the sensations described, he expressed himself as perfectly aware that his sight told him that there was no difference. He converses quite rationally on all subjects and displays no incoherence. His speech is not hesitant, nor is there any tremor in the tongue or lips. There is however, an expression denoting loss of tone in the muscles of the face, as if there were depression of the emotional centres. It may be, however, that this facial expression is partly due, as he says, to anxiety in being placed in

the asylum; his wife and children having no means of subsistence.

He also complains of this curious trickling (formication) coming up his back and over his head and to his forehead, but not on to his face; it travels also down his arms to the hands; he thought it was the medicine that he had in the London Hospital.

October 27, 1901.—Severe girdle sensations, which he describes as if something was squeezing him in a vice.

Patient looks well, good nutrition. He complains that he has had shooting pains in the upper part of the chest two or three months ago, but these have now passed off, and he now feels at night a sense of tightness and difficulty of breathing, with a feeling of soreness. It wakes him up with a struggle to breathe. It catches him in the throat. All over the whole head he has a feeling of tightness. When it comes upon him he feels as though thousands of needles were working upon him. He is obliged to take medicine to keep his bowels open. He complains also of burning feelings both in passing his water and motions. Passes his water pretty freely. No hallucination or delusions; recognised me perfectly. Has nothing to complain about food or treatment.

Speech is not affected. Sways a bit with his eyes shut. Can touch the tip of his nose with forefinger of both hands, has no ataxic gait. He says himself that he has a tendency to fall either one way or the other. Pupils equal, 4 mm., inactive to light, react to accommodation. Some limitation of the field of vision of the right eye in the upper and inner parts. Colour vision of the right eye somewhat considerably impaired, recognised yellow, not red, green or blue. Slight tremor of the tongue, slight tremor of the lips. He has rather a dull, heavy expression. There is a belt of cutaneous anæsthesia corresponding to the third, fourth, fifth, and sixth segments, with some hypalgesia in this region. On the feet and lower part of the legs and over the lower part of thorax and epigastric region there is some delay and hypæsthesia.

March 18, 1902.—The chart of the cutaneous anæsthesia has not appreciably altered. He still suffers with attacks of severe pain, which he says is like electricity; he also suffers with attacks resembling in some respects angina, which Dr. Jones says are of the nature of cardiac crises. The patient described the attacks to me as follows:—"He has always a feeling of a band around the chest, but at times it is as if it were being tightened up, making him unable to breathe, and necessitating him assuming a

sitting-up posture." The feeling of constriction, he says, spreads to his throat as if he were being choked (laryngeal crises?). This sensation is accompanied by severe pains shooting all over his body. He still has the feeling he described on admission as of a trickling of a fluid under his skin spreading up the back of his head over his forehead and on to his face. He believes that this feeling is associated with a swelling of the cheeks. Mentally he has not changed, there is no sign of dementia.

Case 28.—*Tabes of sixteen years' duration, commencing with optic atrophy, followed soon after by mania with delusions of persecution; subsidence of acute mental symptoms and gradual development of spinal symptoms, which after ten years led to helpless ataxy, mental enfeeblement, but no progressive dementia. Death from acute dysentery. Tabic lesion of spinal cord and roots, in cervical and lumbo-sacral regions, especially; affection of both exogenous and endogenous posterior spinal systems; marked patchy pia-arachnoid thickening over prefrontal, frontal, and central convolutions; chronic atrophy of superficial layers of fibres and cells of cortex in these regions without vascular changes. Was this a case of mania and tabes, or tabetic general paralysis with arrest of mental symptoms?*

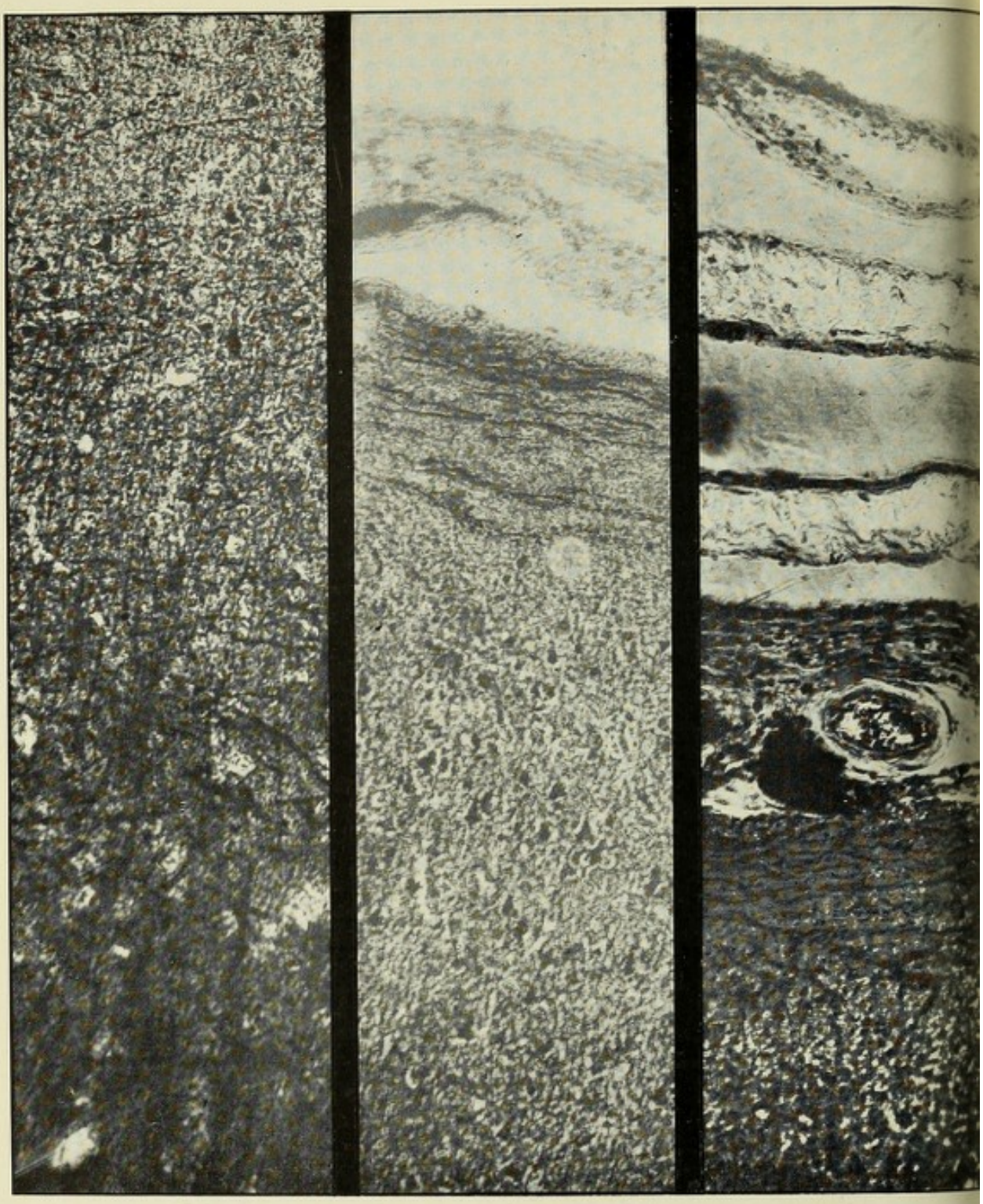
C. R., aged 38, admitted to Colney Hatch in 1886 for mania and delusions of persecution. He had lost right eye when a youth as a result of injury. Some months before admission to asylum the sight in the left eye began to become dim, and he rapidly became completely blind. By occupation a colourman, the question of lead-poisoning was considered as a cause of his affection. There was no history of syphilis obtained, nor were there any signs on the body. The cause of the attack of mania was associated by his friends with the worry caused by the loss of sight. While in the asylum and after the subsidence of the attack of mania, ataxic symptoms developed, and in 1897 I first saw him in good physical condition, although he was in the third helpless stage of ataxy. He was unable to stand without assistance, and had to be supported by two attendants when he walked. All the deep reflexes were lost. The plantar reflexes were lost, the gluteal present on the right, absent on left, epigastric reflexes increased; cremasteric absent. The muscles are well developed, and the muscular strength is good; there is considerable hypotonus in hamstring muscles, the limb can he raised when extended at the knee to a right angle with the body.

Sensation.—He does not complain of pains, numbness, or formication. There is diminution of sensation and delay to all forms of stimulation, with very incorrect localisation in the feet and legs. Over the abdomen and thorax there is apparently some hyperæsthesia, especially to cold, but no anæsthesia or analgesia. He has no loss of sensation, painful or light tactile in the tips of the fingers, but there is a loss of sense of position in the joints. There is marked loss of sense of position of the joints of the lower extremities. The left pupil is dilated, and reacts neither to accommodation nor light. There is well-marked primary atrophy, with cupping of the disc.

Visceral.—He is unable to retain his water which dribbles away from him; there is no history of gastric or other visceral trouble.

Mental.—He is somewhat weak-minded, but will converse about general topics, remembering the visits of his friends and what they said, and the day of the month. His comprehension is fair and he answers questions rationally; occasionally he is subject to violent outbursts of temper, but he has no delusions or hallucinations. There is continually twitching of the facial muscles and of the left orbicularis palpebrarum. He has no hesitancy or slurring of speech and the dementia has not been progressive.

The patient remained in *statu quo* until an outbreak of dysentery occurred in his ward; he was sick, took to his bed and passed a large quantity of blood and slime, became very anæmic and collapsed and died in two days. At the *post mortem*, I found the whole of the large bowel and the lower twelve inches of the small bowel filled with blood and slime; there was no ulceration anywhere in the alimentary canal to account for this; the mucosa and sub-mucosa were greatly swollen and congested and, microscopically examined, showed the characteristics of the very acute fatal form of dysentery. The other thoracic and abdominal organs were healthy with the exception of some atheroma of the aorta. The brain weighed 46 ozs., there was not much sub-arachnoid fluid, but there was considerable pia-arachnoid thickening over the frontal and central convolutions on both sides. There was especially thickening of the membranes and atrophy of the superior parietal lobe, just behind the upper fourth of the ascending parietal, and on stripping the membranes, a shallow pit, 1 in. square was seen, due to this atrophy, symmetrical in both hemispheres. The lateral ventricles are faintly granular and not dilated; the fourth ventricle is somewhat dilated and



(*C*) (*B*) (*A*)

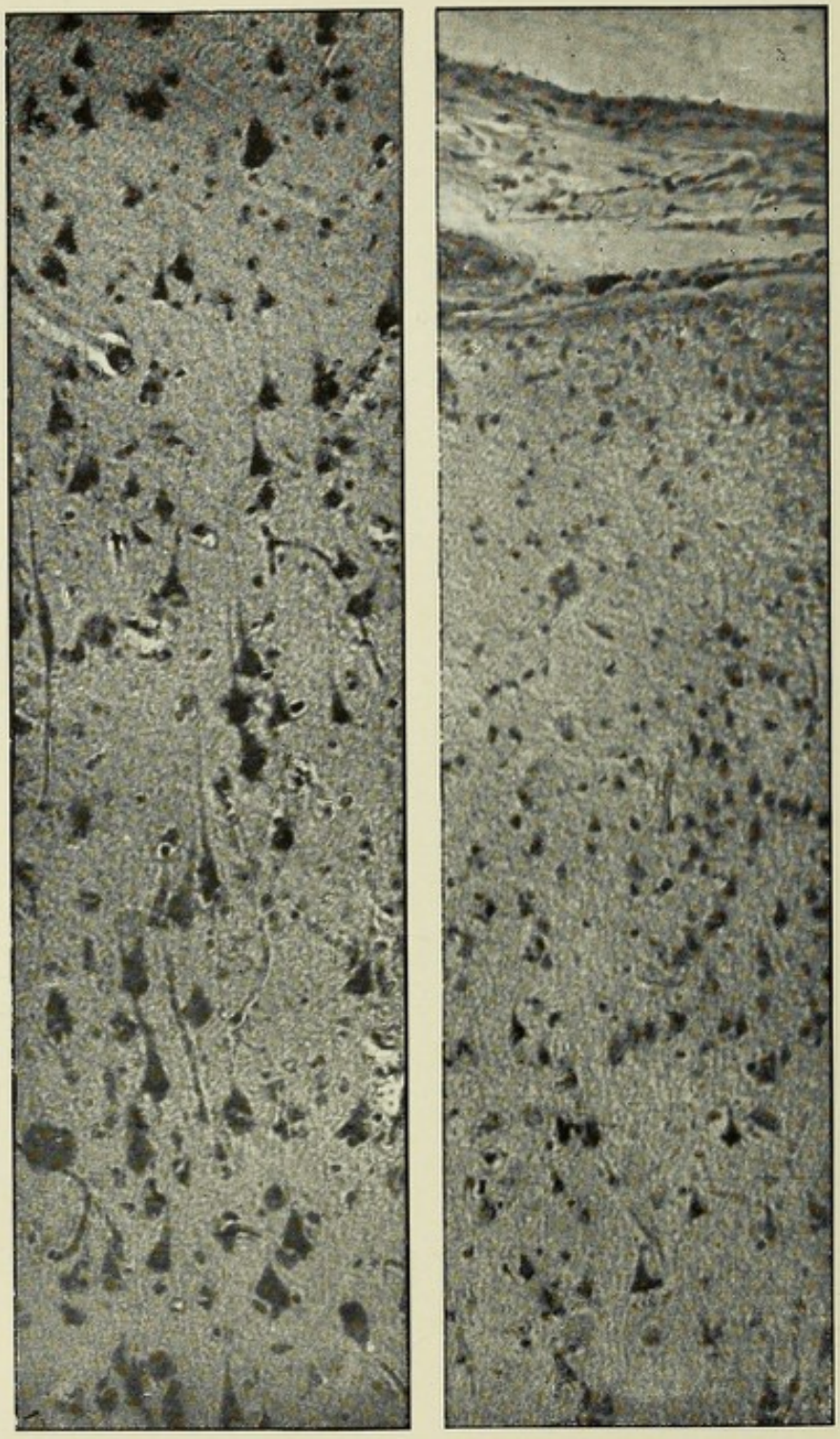
(*A*) Thick section of cortex (ascending parietal) with adherent pia-arachnoid which is gre thickened, and consisting in this situation of a series of strata of dense fibro-vascular tissue. tangential and supraradial fibres in the subjacent cortex are diminished considerably, but section is too dense to show this properly.

(*B*) Thinner section of cortex (ascending frontal). Membranes not so much thickened, a number of tangential fibres seen.

(*C*) Deeper layers of cortex (ascending parietal) showing radial, supraradial, and interra fibres.

Magnification 150 diameters.

PLATE I.



(*B*) (*A*)

Section of superficial layers of ascending frontal, showing (*A*) Molecular layer and small pyramids; (*B*) medium sized pyramids. Magnification 180 diameters.

To face p. 125.

there are some granulations. There is slight thickening of the pia-arachnoid over the superior temporal convolutions and along the tip of the temporal lobe, and the anterior part of the uncinate convolutions. The thickening of the membranes and atrophy of the convolutions is more apparent in the upper two-thirds than the lower third of the central convolutions.

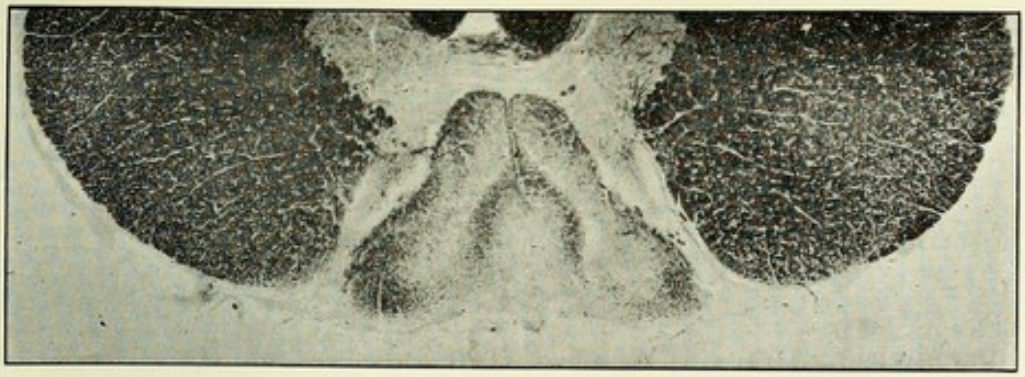
Preliminary microscopic examination of the brain by Nissl and Weigert methods of staining cells and fibres showed the following conditions :—The small cells of the molecular layer of Cajal are greatly diminished in places ; also, but to a less degree the small pyramids, and less still the medium sized pyramids. Those cells that remain of the pyramidal layers have a fairly normal appearance and are arranged in columns with straight apical processes which can be followed for some distance. The large Betz cells lying in small groups of four to six are for the most part quite normal, they have all their processes, and the majority of cells present no chromophilous changes. Portions of the first frontal, ascending frontal, and ascending parietal, stained by Wolter's method for fibres show thickening of the pia-arachnoid of considerable degree and proportional to an atrophy of the tangential and supra radial and interradial fibres, quite as marked as in a case of fairly advanced general paralysis. There are no vascular changes, no plasma cells, and comparatively little glia cell proliferation (*vide* photo-micrographs, Plates I. and II.) The complete examination of this brain will occupy the subject of a future communication, being one of considerable interest. Is this a wasting of physiologically correlated structures in a case of tabes, or is it the lesion of a case of arrested general paralysis ? When such difficulties of diagnosis exist, as in this case, one is impressed by the unity of the two diseases.

Microscopical examination of the spinal cord.—The degeneration in the spinal cord corresponded fairly accurately with the clinical symptoms observed during life. The cord was, with the exception of the posterior columns, of normal size, the myelin sheaths, unlike many tabo-paralytic cases, stained remarkably well. There was no degeneration of the pyramidal systems of fibres to be found anywhere. The atrophy was limited to the posterior roots and posterior columns of the cord. There is almost complete disappearance of the fibres of lumbo-sacral roots forming the cauda equina. The upper four lumbar and the last four dorsal roots at attachment to the cord possess still a large number of healthy fibres ; in the mid and upper dorsal roots the fibres become less and less numerous until the cervical region is reached. At the

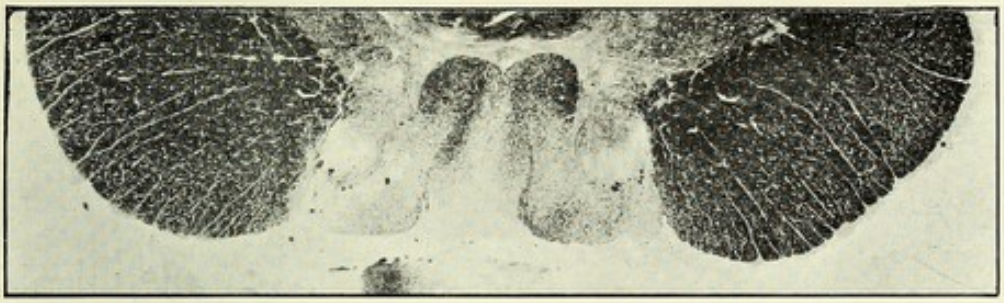
seventh cervical there is a great increase in the number of fibres, and above this level the number is normal, and there is abundance of fibres in the cornu radicular zone.

Spinal cord.—The column of Goll is completely denuded of fibres in the cervical region, except a Λ shaped portion in front, which corresponds to the lower dorsal and upper lumbar roots; in front of this area of fibres there is a Λ shaped area of sclerosis which corresponds to the outfall of fibres in the upper dorsal and lowest cervical roots. Examination of each segment of the cord shows that this most ventrally placed Λ shaped area of sclerosis corresponds to the outfall of fibres coming from the upper three dorsal and last cervical roots. These represent fibres which carry impulses from the deep structures, muscles, and joints of the hand. Thus at the eighth cervical we find a complete atrophy of entering root-fibres. The Λ shaped area of healthy fibres between the two areas of sclerosis are partly descending endogenous fibres, for fibres can be seen passing obliquely from the cornu commissural zone into this area, forming below the comma shaped tract. A good number of the fibres may, however, represent long fibres from roots of the lowest dorsal and upper lumbar, seeing that the clinical signs and microscopical examination showed these roots were not much affected. In the lumbo-sacral region there is much shrinking of the posterior columns; the only fibres that exist are the endogenous occupying the cornu-commissural zone, oval area, septo-marginal and Gombault's triangle, but the fibres in these tracts appear very much diminished. The fibres which are most atrophied are those forming Goll's column (long fibres), cerebral, and those concerned with reflex spinal tonus, viz., those occupying Charcot's root zone. These two sets of fibres seem very completely atrophied. The fibres which enter the column of Clarke as soon as it appears at the level of the second lumbar segment, are not completely destroyed; some fibres can still be seen entering the column at different levels, and breaking up into a plexus around the cells; still there is very considerable atrophy of this plexus (examination of the photomicrographs of Plate III. will enable the reader to follow the above description of the changes in the spinal cord). Lissauer's tract is not so markedly atrophied even in the lumbo-sacral region. This may account for the fact that the cutaneous sensory disturbances were in proportion to the marked ataxy and helplessness, but slight comparatively. The slow progress of the disease may be connected with the blindness from optic atrophy. Examination by Nissl's method showed that

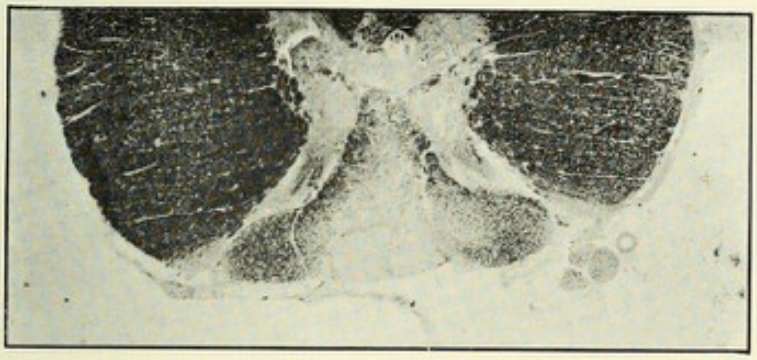
PLATE III.



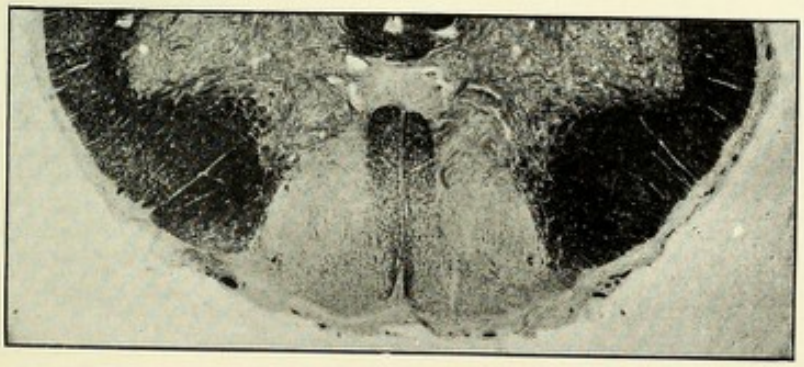
Fourth Cervical.



Eighth Cervical.



Tenth Dorsal.



Fifth Lumbar.

TABES IN ASYLUM AND HOSPITAL PRACTICE.

To face p. 126.

the spinal motor cells were normal in appearance, the granules of Nissl were normal in the cytoplasm and on the processes. This agrees with the fact that there was not very marked hyptonus, and the muscles were of good colour and in no degree wasted. There was an abundance of small association and sensory cells of normal appearance in the anterior and posterior horns.

Case 29.—*Advanced tabes (which commenced with gastric crises at* 17?), *ataxy and double vision at* 28. *At* 39 *admitted to asylum for an attack of mania with visual hallucinations and delusions.*

A. R., aged 39, general servant. No history or signs of specific disease. She says she had rheumatic fever at 17 (?) No history of ulcers on the eyes. Just after age of 17 she had attacks of vomiting, which would come on without any food in the stomach. Mother died of consumption; father of senile decay; nine brothers and sisters, of whom seven are alive. At the age of 26 she commenced to have shooting pains in the legs. Could not walk about in the dusk without tottering, and used to totter when she washed her face. At 29 she had double vision, and saw Mr. Nettleship, at Moorfields. He said that there was loss of sense of position in the feet; she was taken into St. Thomas's Hospital under his care. She had drooping of the right eye, and was treated with blisters round the forehead. She went out and took a situation, but found that her right leg used to give way from time to time; she still had gastric crises; gradually walking became more difficult, and a stamping gait developed. She went into Highgate Infirmary incapacitated; gradually legs became more helpless, and the vomiting became very severe. The vomiting has ceased for the last two years, but she still has the girdle and lightning pains, bnt not nearly so bad as before. She has not full control over the bladder, fairly good control over the bowels, but cannot wait for either micturition or defæcation. She had been suffering from irregular catamenia, which sometimes was suppressed for a year; this came on before the diplopia. Had complete loss of power in the legs for six years. Prior to this there were spontaneous unconscious irregular jerky movements when she was in bed. She also had irregular jerky movements in the hand, in attacks resembling St. Vitus' dance. Had pins and needles in the fingers (paræsthesia).

In August, 1899, she suddenly lost herself, and developed a shakiness in the hands; she spilt her food owing to choreiform spasm. She lost consciousness, and had visual hallucinations.

She thought she saw the matron making preparations to burn her up, and this became a fixed idea for a short time. She did not remember her relatives when they came to see her. Delusions that she was being tried by a judge and jury for some supposed crime. She had the delusion that her brother had been killed in trying to assist her. She was transferred to an asylum at the end of October. When, however, she was admitted to Colney Hatch, there was but little mental affection. The nurse says she has had no delusions or hallucinations. She now seemed very intelligent, and gives a most intelligent history of her illness.

Condition on admission.—Loss of sense of position in both hands. Cannot touch nose with fingers when the eyes are shut. No affection of speech ; hearing normal. Legs extended in bed, feet inverted and flexed at the ankle, the knees extended. Complete paralysis of the feet and legs ; she is unable to move any of the joints of the foot or leg, and cannot move her hips. There is marked foot drop and inversion of the feet. Knee-jerks absent on both sides ; the muscles are wasted, but how far this is due to prolonged disuse I cannot say. Plantar reflexes gone, epigastric reflexes brisk. Great weakness in muscles of back and neck, causing a certain amount of lordosis. She has to jerk her head to get it erect. All the muscles respond normally to the faradic current. No tenderness on percussion over her spine. Right disc indistinct, and smaller than natural. Pupils, 6 mm., react to accommodation, but not to light. The sensory charts made in this case exhibited very marked cutaneous sensory disturbances to touch, pain, and heat and cold, in the regions supplied by the roots of the third to the tenth thoracic, and from the fourth lumbar to the last coccygeal. There is delay and diminution of cutaneous sensibility of a partial character in the areas supplied by intervening roots, and above the complete anæsthesia of the trunk there is hypæsthesia of the skin of the arms and of the trunk as high as the second intercostal. All the roots, therefore, below the third or fourth cervical are affected, and one would expect to find complete denudation of fibres in the roots, corresponding to the areas of complete anæsthesia, accounting in a measure for the complete loss of power in the lower extremities.

Case 30.—*Tabes, married woman, syphilitic history, mental crises in the form of hallucinations and delusions, Charcot's joint, gastric crises, preceded by pains in the swollen knee-joint.*

E. C., aged 39, married woman admitted to Banstead, August, 1898, with acute mania. hallucinations and delusions. There was

a history of six miscarriages and no children, but no history of insanity or nervous disease in the family. There are scars of old syphilitic ulcers on the legs. The symptoms of tabes commenced at the age of 33 with lightning pains, for which she attended St. Thomas's Hospital. When she was 36 years of age she fell down stairs, her right knee swelled up, and she was *delirious*. She was taken to St. Thomas's Hospital, but was a week later transferred to the Lambeth Infirmary on account of hallucinations and delusions. "Men with monkey's skins came and stole her things out of the drawers." It was real to her then, but she now knows it was only a delusion. Once only while she has been at Banstead (two years), has she had any real mental disturbance. She was, according to the nurse, delirious and had visual hallucinations: she saw her husband and friends at her bedside, and wanted to get out of bed and go with them. She suffers with severe attacks of gastric crises at times, which last for several days. The crises seem to be preceded by pains in the right swollen and diseased knee-joint. Over the external and internal condyles there is a sinus, from which pus is constantly discharging. The joint is nearly 20 ins. in circumference and disorganised. The muscles of the leg are greatly wasted, and there is well marked foot drop. The patient states very definitely that during the gastric crises lightning pains radiate all over the body, passing down the legs and arms, up the back, and terminating in the forehead, but never spreading to the face. The knee-jerk in the left leg is absent; there is hypotonus of the hamstring muscles, and a marked loss of joint sensibility in the lower limbs. Argyll-Robertson pupils. There is no loss of tactile or painful sensibility in the upper limbs, but slight incoordination and loss of sense of position. Painful and light tactile sensation is lost or impaired below the knee, and there is slight blunting or loss of light tactile sensibility in the area corresponding to the sixth, seventh and eighth segments. There is no bladder or rectal trouble. The patient was sent to St. Bartholomew's, and the right leg amputated above the diseased joint, which was sent to me for examination, by the kindness of Dr. Claye Shaw and Mr. Walsham.

Examination of the nerves of this joint showed atrophic degeneration somewhat similar to that which is found in the posterior root of an ataxic case, viz., disappearance of myelin from sheath, proliferation of nuclei of primitive sheath. This, of course, may be simply a disuse atrophy associated with prolonged immobility, or it might be explained by damage of the nerves from the dead bone and discharging sinus.

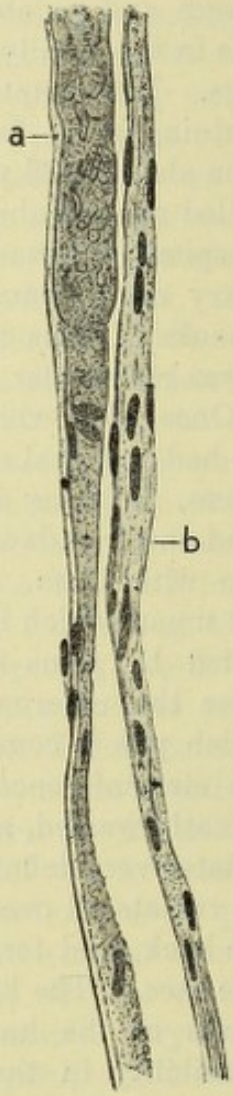


FIG. 19.

Teased nerve-fibres entering the peroneus longus muscle away from any obvious septic inflammation. At (*a*) the myelin is shown filling the neurilemma; it has an opaque dull appearance, indicating a congulation neurosis. At those portions of the fibre where the myelin has disappeared the nuclei have undergone proliferation; (*b*) is a fibre in which the myelin has quite disappeared. Staining, alum hæmatoxylin, mounted in ammonium picrate and glycerine. Magnification 450 diameters.

GROUP 3.—Tabo-Paralysis.

Case 31.—*Onset with fits, headache, loss of memory, insomnia; admitted to asylum with symptoms of acute mania and physical signs of tabes. Subsidence of mania, marked ataxy, convulsive seizures, gastric crises, shooting pains with associated delusions, lucid intervals, but a progressive mental enfeeblement. Death.*

I. D., female, aged 31, single. Occupation, colour printer. Admitted to Cane Hill on October 2, 1897.

History.—No family history of insanity or phthisis. She attended a dispensary about seven months for some disease of the genito-urinary organs, but whether this was syphilis cannot be definitely ascertained. She had two fits five months ago, and for the last three months she has had headache, loss of sleep, and failure of memory. Quite recently her speech changed, and she became very restless and talked incessantly. The cause of the attack is stated to be disappointment in love.

On admission.—A young woman, thin and badly nourished, but with no signs of organic disease of viscera. Tongue clean, pupils equal and contracted. No reaction to light or accommodation. Knee-jerks absent. She has general movement of head and arms. Waves her arms continually and moves her head about in all directions. The movements of her arms are purposeful. She undoes her hair and does it up again continually, and keeps unbuttoning her dress. Her gait is unsteady and ataxic. She is destructive and very mischievous. Is wet and dirty in her habits. Will not keep in bed, but rolls off on to the floor. Mentally she is in a condition of acute mania. Is very restless and resistive to everything. Unable to answer the simplest questions properly, but rambles off irrelevantly. Childish and foolish. Memory bad.

November 2.—Symptoms of mania persist; she has required sedatives (such as chloral and paraldehyde) on account of her extreme restlessness. Still wet and dirty and very destructive.

December 1, 1897.—She has become much clearer mentally. The attack of mania has subsided. She is fatter and stronger, and the movements have entirely ceased. She now has well-marked signs of tabes dorsalis. Ataxic gait and loss of knee-jerks. Pin-point pupils, slight reaction to accommodation, but not to light. Has anæsthesia of the ring and little fingers on both sides.

January 2, 1898.—She has lately been having attacks of vomiting, and has complained of pains in her abdomen and legs. Is very shaky and ataxic, and is becoming very feeble. To-day, she had a convulsive seizure, and became comatose for some time. Her face has lately become greasy and expressionless.

March 1, 1898.—She has been practically bedridden since last note. Quite helpless and feeble, and has become wet and dirty in her habits again. Often vomits and complains of pain round her waist. Her speech is now slurred, and her tongue tremulous, and she is more demented. Her condition now differs in no way from the last stage of general paralysis.

April 14, 1898.—Still bed-ridden and extremely feeble and

helpless. Has become very rigid in muscles of neck and limbs. Is quite demented and cannot understand or answer the simplest questions. Has paresis of left internal rectus. Occasional pyrexia. Her right pupil is now slightly larger than the left, but there is no reaction to light.

May 18, 1898.—Still in bed. General rigidity of muscles all over body. Wet continually. No vomiting now. She improved in mental condition, and was able to get up and sew a little, and take a fairly bright and intelligent interest in her surroundings; but in the middle of July, she became very restless and excited, refusing to stay in bed because she said mice ran over her body and pillow. She makes frequent attempts to catch them. Continual use of sedatives necessary on account of her restless condition. She complains of the treatment by the nurses, that they had put something chopped up in her milk, that they had scraped her bowels out and spoiled her features. This was no doubt an insane interpretation of the gastric crises. (F. W. M.).

August 12, 1898 (F. W. M.).—The patient says she is feeling much better. The nurse says she is quiet now, but she has been very excited and troublesome. There is typical ataxy of the lower limbs. The knee-jerks are absent and she is unable to stand or walk alone. When supported by two nurses she can stand with difficulty, but on closing the eyes she would fall if not supported. She can touch the tip of her nose with her finger, and she can write her name, but there is a good deal of fine tremor in the strokes. She complains of sharp shooting pains in the legs and round the stomach. She passes water involuntarily, but has control over the bowels. She has not menstruated since she has been in the asylum. She has lately had an ulcer on the cornea. Pupils, Argyll-Robertson, small, equal. There are no synæchiæ, and the fundi are normal. She complains of a feeling like wool on the soles of her feet, and she is unable to say what is being done to her great toe when it is flexed or extended. There is some loss of painful and tactile sensibility in the lower limbs. There is no tremor of the face or tongue. There is slight ptosis of the right eye-lid. Smell and taste are good, also hearing. Speech normal.

Mental condition.—She appears to me quite rational, but her memory is somewhat defective. She told the nurse that she had a baby at Liverpool, and that she has had two abortions. She tells me the same story, and that she has lived with several men. Some time ago she had delusions that there were mice in the bed. When I last saw her she was in an unconscious state, and I

judged that she was suffering from a congestive seizure of general paralysis. (F. W. M.)

June 19, 1899.—Mentally the patient has greatly improved since I last saw her. She talks quite rationally, has no loss of knowledge of time or place, her speech is unaffected. She tells me that she has had many attacks of vomiting lately, and diarrhœa. She complains also of a tight feeling in her left side (gastric crises). She also tells me that she has a feeling of wool on the soles of her feet. There is considerable blunting of sensation in the lower extremities, especially on the left side. She feels sharp pricking, but there is considerable delay, and blunting of sensation. There is no incoordination in the hands now. She can knit and sew. Complains of dimness of vision in the left eye.

October, 1899.—The patient has been free from mental symptoms for the last six months, and she talks in a perfectly rational manner, without any hesitation or slurring of speech. She complains of a girdle sensation round the waist, and for the last three months she has suffered from severe attacks of vomiting and nausea. She has had shooting pains in the legs, is constipated, and wets the bed at night; she has had no more fits. There is considerable incoordination in the hands, and she is unable to sew as well as when I last saw her; she has numbness of the ring and little fingers of the left hand.

October 9, 1900.—Patient is in a fairly rational condition of mind, and able to answer questions. She knew me quite well. She has had many attacks of vomiting since I saw her, with shooting pains in the left side and legs. She has attacks of pain in her legs nearly every day. The attendant informs me that not long ago she became very restless and violent, and very difficult to manage. She believed that she was going to marry Dr. B., and wrote numerous letters on the subject asking him to provide clothing for her. She tried to tear out all her gray hairs, and wished to have all her teeth drawn in order that she might be made beautiful and captivate the doctor. She was very destructive, tearing up her clothes. She remained in this maniacal state for over a month. When she had fits she would lose consciousness, sometimes for four hours together; the convulsions were mostly on the left side.

Physical examination.—The limbs are well nourished, but there is very marked hypotonus in the hamstring muscles. The extended limbs can be bent to an angle of 75° with the body. There is loss of sense of position in the hands. Sensation, tactile, absent in the shaded parts of the diagram, although there are

scattered patches of normal sensation with a delay in response, tested by lightly touching with the finger. Painful sensation, pricking with a needle; result shown in the accompanying diagram, likewise thermal sensation. In parts of the body other than those indicated in the diagram she always gave a correct answer, showing that she was able to appreciate and respond to the stimulus.

July, 1901.—Smell and taste.—She cannot smell peppermint, assafœtida, or rose-water. She can taste quinine, but syrup and acids are not appreciated. Hearing.—Hears a watch 18 inches

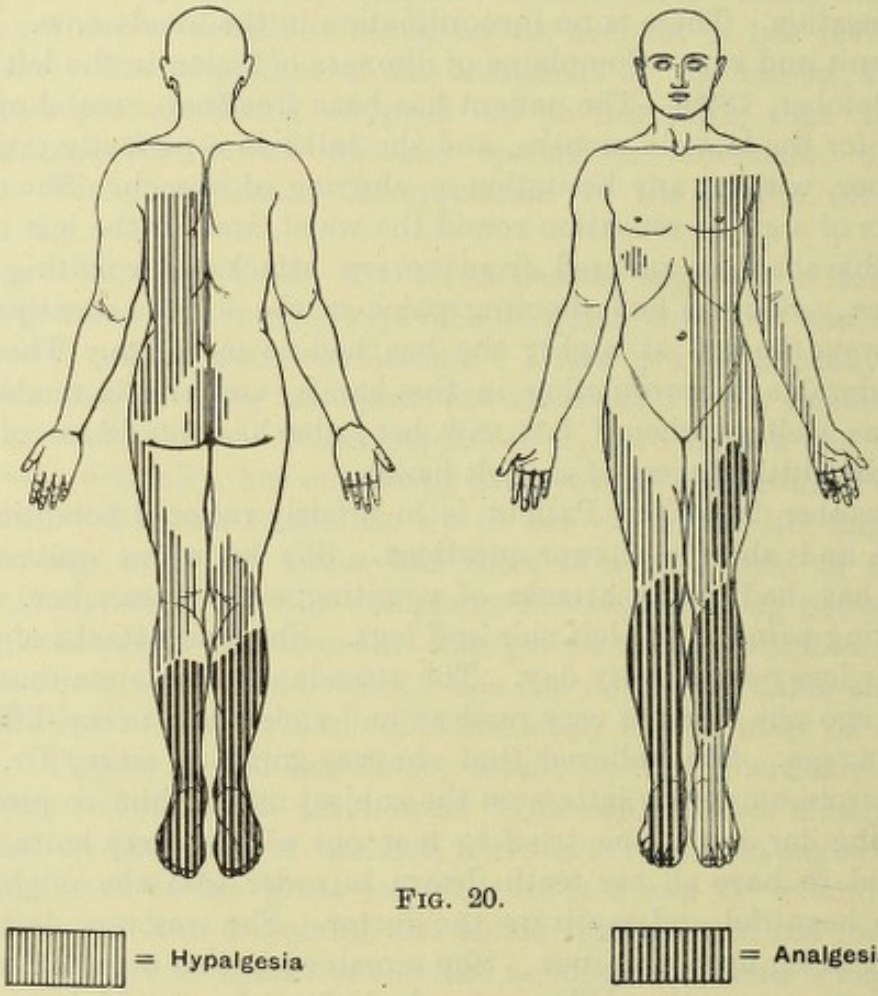


FIG. 20.

on either side. Colour vision.—Picks up correctly colours with one eye closed, but marked incoordination and failure of judgment of distance noticed. Pupils.—Right 3 mm., left $2\frac{1}{2}$ mm.; inactive to light, very sluggish to accommodation, outline irregular. She takes her food, but is much weaker than she was; she has had no fits lately and no sickness. Tongue and lips tremulous, but the speech is not affected. She has no control

over the sphincters, and when she wets the bed she says that some one else has done it. Although so emaciated, she has a ravenous appetite. After she has eaten her dinner, she fancies that some one else has had it, and she will steal another patient's dinner if she is not looked after. She is quarrelsome, and will smack the faces of patients who sit next to her. She forgets the incident the next minute. She forgets the names of people, but she has not personal illusions. She has not complained of bad tastes in the mouth or bad smells, nor has she now any delusions

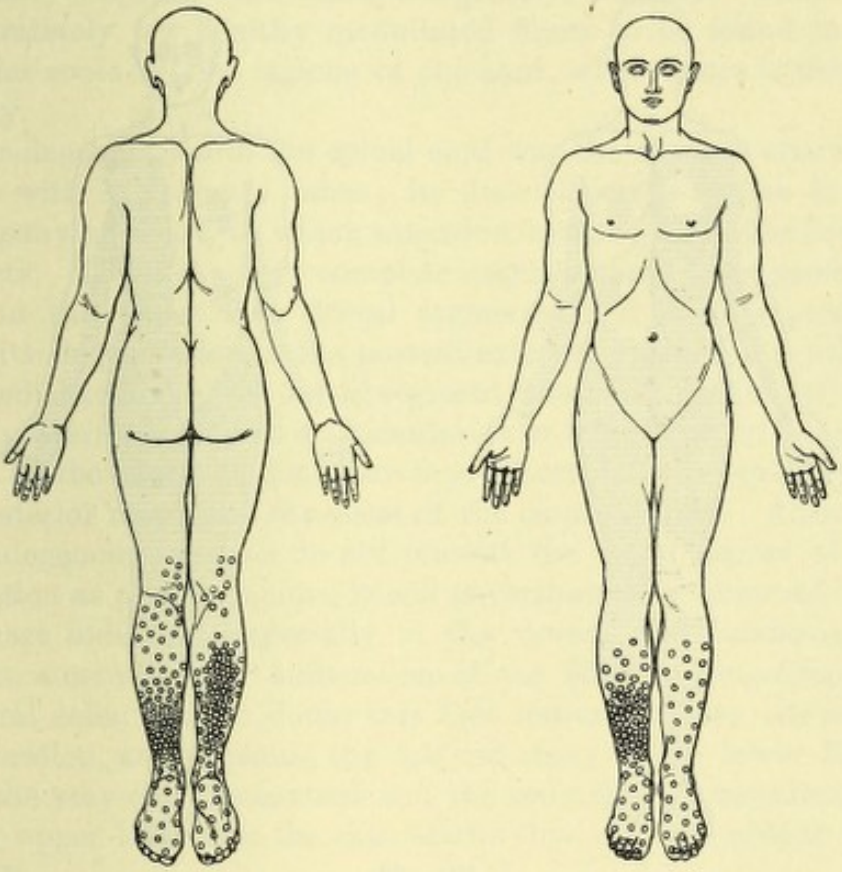


FIG. 21.

= Thermo-anæsthesia

of poisoning. She recognised her friends, but her knowledge of time and place are extremely defective. She does not know whether it is winter or summer, morning or afternoon. I told her that she had had her dinner, she then replied that it must be afternoon.

January, 1902.—Death from pneumonia.

Autopsy.—Broncho-pneumonia affecting the right lung especially. Nothing especially noteworthy in the other organs, except

the ovaries; the left Fallopian tube is matted to the ovary and uterus, and exhibits the appearance of old non-tubercular salpingitis. There were no signs of syphilis on the body, but the vagina was large, and this, together with the absence of hymen, a scar on the cervix and the condition of the Fallopian tube mentioned, accords well with her statement of abortions and the history of some venereal infection. *Brain.*—No adhesions of the dura mater, slight excess of sub-arachnoid fluid, slight thickening

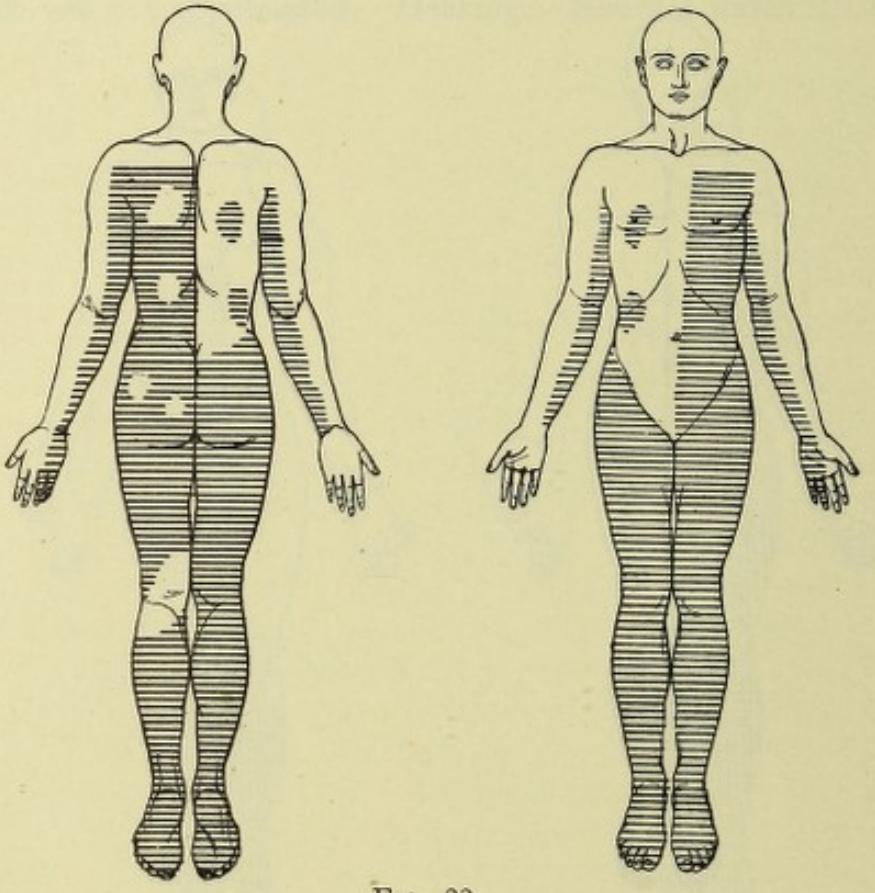


FIG. 22.

= Cutaneous Anæsthesia

and opalescence of pia-arachnoid over the frontal and central convolutions. Some adhesions along mesial surface of frontal lobes, very little wasting or thickening of membranes of pre-frontal or orbital lobes. Some wasting of first, second, and third frontal at junction with anterior frontal; no obvious wasting of Broca's convolution; optic nerves small, but not gray. Olfactory nerves small, membranes thickened, opalescent, and adherent about the tip of the temporal lobes and origin of olfactory. Granulation of

lateral ventricles, but very little dilatation. Marked granulation and some dilatation of the fourth ventricle; erosions of central convolutions on stripping the membranes, decided atrophy of upper two-thirds of central convolutions on both sides. Naked eye atrophy and gray degeneration of the posterior roots and posterior columns of the spinal cord.

Microscopical examination.—Teased preparations of posterior roots exhibited a large number of empty sheaths with proliferated neurilemmal cells; others, the myelin sheath partially gone, or irregularly disposed—indicating a regressive atrophy. There are comparatively few healthy medullated fibres to be found in the posterior roots of the regions of the cord, where there is usually atrophy.

The degeneration of the spinal cord was the same in character as met with in ordinary tabes; its distribution is shown in the accompanying chart, to which attention is called, as to the following facts. There is a very complete degeneration of the posterior roots in the upper nine dorsal segments, and a corresponding extensive degeneration in the postero-external columns; it will be observed that at the fifth dorsal segment, there is a marked atrophy of the posterior horn, and an indentation in this region on one side. In the lumbo-sacral region there is also complete degeneration of the posterior roots, and the roots of the cauda equina. Although the endogenous systems do not present the same degree of degeneration as the exogenous, it will nevertheless be observed that the chart indicates, especially in the dorsal and lumbo-sacral regions, a considerable destruction of the fibres arising from the cordonal cells, and no doubt this fact indicates more advanced degeneration, and explains the marked ataxy of the lower limbs from the very commencement and the only slight incoordination of the upper limbs, for the case shows that she was able to knit and sew.

The distribution of the degeneration accords fairly well with the chart (fig. 23), indicating the distribution of anæsthesia and analgesia observed during life, six months before she died. Probably the roots on the right side were subsequently affected, for the roots in the dorsal, lower lumbar and sacral regions were practically denuded of fibres on both sides.

The whole cord was not larger than that of a child of two years, particularly is this noticeable in the dorsal region. Another fact indicating a metabolic change was the difficulty with which the myelin sheaths of the nerves stained, resembling in this respect the cords of new born children. Owing to the extreme atrophy of the posterior roots, therefore, of the projections in the

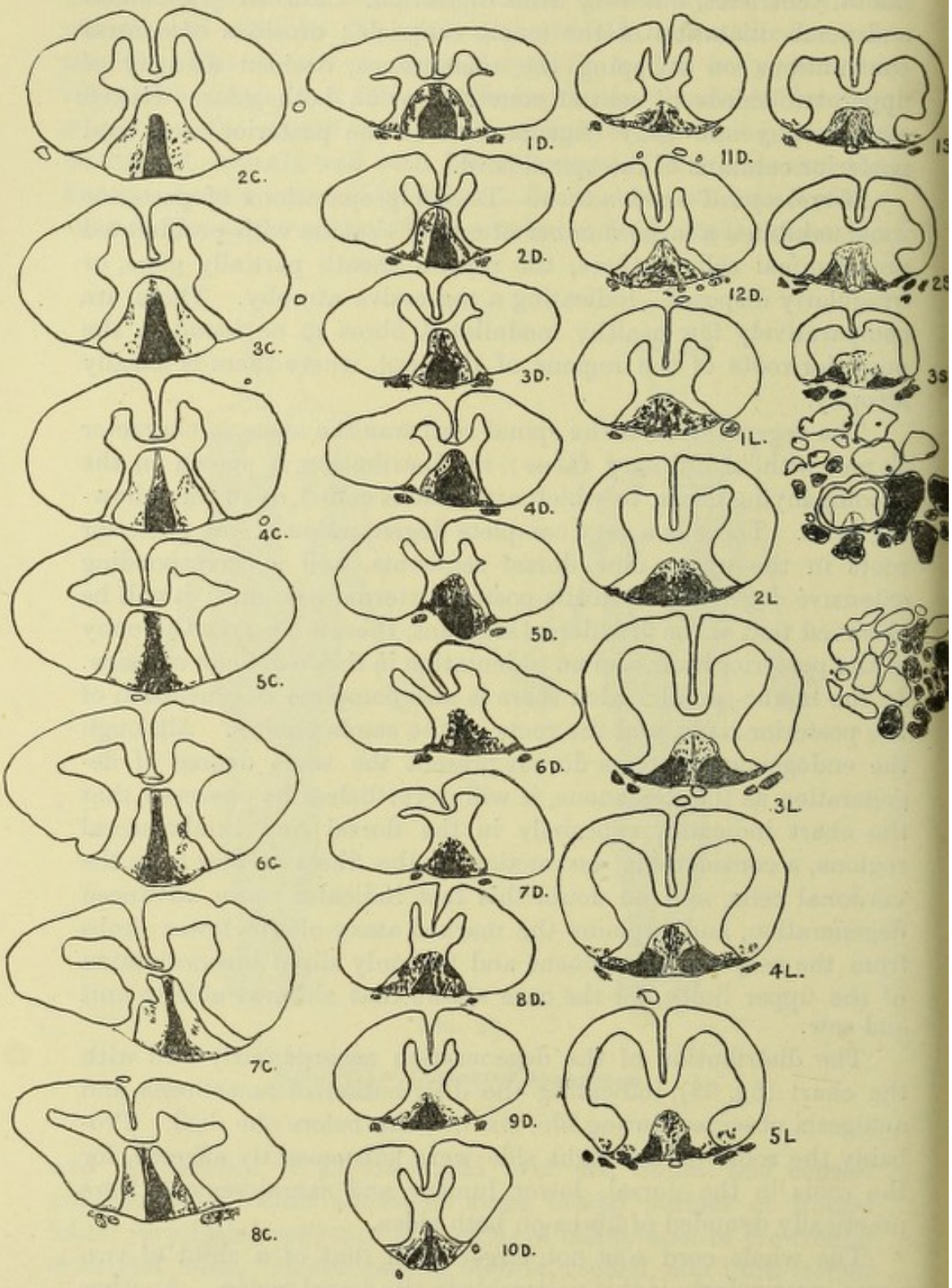


FIG. 23.

Diagram made with Edinger's projection apparatus, from sections of the spinal cord stained by Weigert-Pal method. Complete degeneration and atrophy of fibres indicated black, partial atrophy by black dots.

cord, the course of endogenous fibres still existent could easily be followed and the following notes were made (*vide* fig. 24) :—

Cauda equina.—Complete degeneration of all the fibres of the posterior roots, with imperfect staining of fibres of anterior roots. A section of the fourth sacral segment roots included showed a small triangle of fibres in the posterior column, endogenous, and corresponding to the posterior median triangle of Gombault and Philippe. This tract of fibres is continuous with a tract which lies along the median fissure in the first, second, and third sacral segments; at the fifth lumbar segment, there are no root-fibres, and these septo-marginal fibres can be traced along the posterior column to the posterior horn, while other fibres can be traced from the cornu commissural zone to the base of the posterior horn. The septo-marginal tract is seen to be continuous with the oval area of Flechsig, so that we may consider these three tracts with separate names are merely divisions of the same system of endogenous fibres. At the level of the first lumbar and last dorsal, at the very extremity of the posterior median fissure, there exists a small triangle containing still a number of undegenerated fibres. This tract of fibres is continuous below with the oval area of Flechsig, and above with the internal zone, which consists in a great measure of descending endogenous fibres; it is is continuous above in the upper dorsal region* with the comma shaped tract. Study of the sections shows that a large number of reflex collaterals are given off by these endogenous systems and that they consist of two sets of fibres, *ascending* and *descending*. The comma tract, posterior internal triangle, fibres continuous from there along to the median fissure which form the septo-marginal tract, oval area of Flechsig and the posterior triangle of Philippe constitute the latter. Collaterals are given off all the way down in two situations: (1) in the substantia gelatinosa, where the fine fibres of Lissauer's zone end ; (2) in the posterior horn opposite the root zone. The ascending endogenous system forms the cornu commissural zone of Westphal; the fibres from this zone in the dorsal region can be seen very distinctly running forwards in three groups of fibres: (*a*) External to Clarke's column; (*b*) Internal to Clarke's column, to form a plexus at the junction of the anterior and posterior horns; (*c*) Fibres decussating in the posterior commissure from the other side (*vide* fig. 27). These fibres can be distinctly traced, owing to the complete atrophy of the collaterals of the exogenous fibres around the cells of Clarke's column; the fibres from the latter entering the direct cerebellar tract can also be seen distinctly as they pass outwards and upwards across the base of

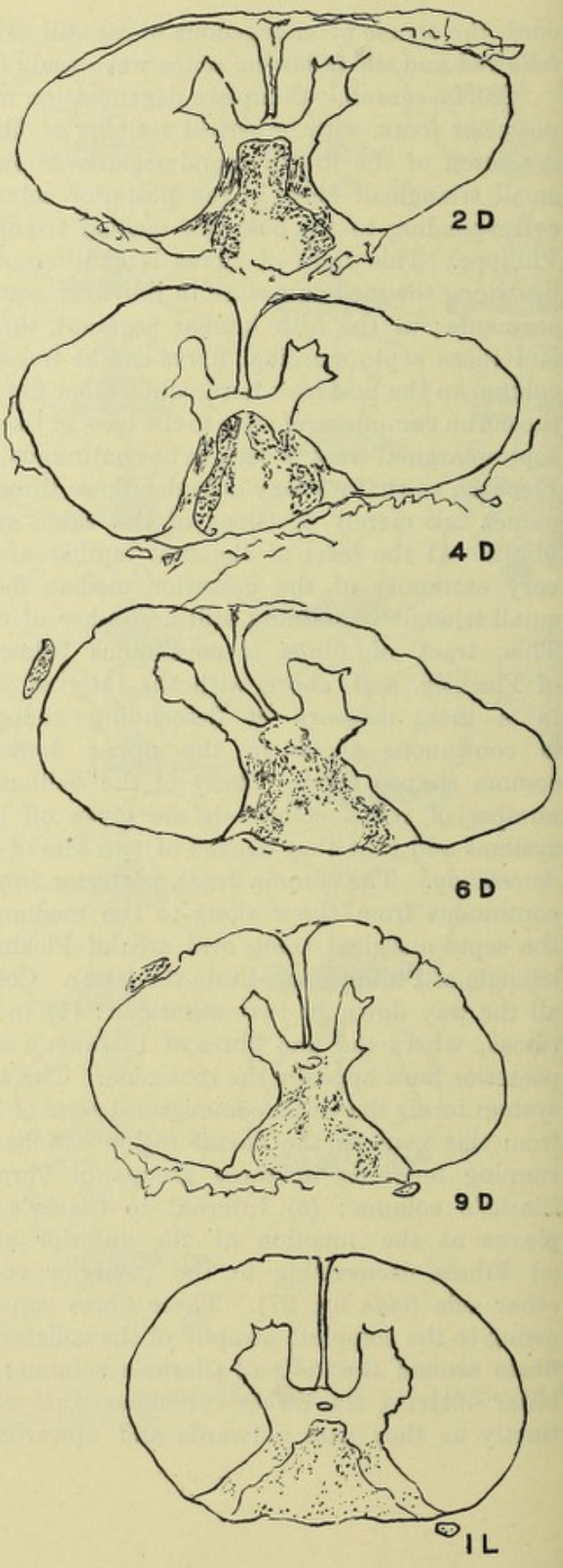


FIG. 24.

Outline drawing of spinal segments, showing fibres of endogenous origin remaining in posterior columns of spinal cord at levels where there was complete degenerative atrophy of the roots 2 D, 4 D, 6 D, and those intervening; also nearly complete atrophy at lower levels in the dorsal region. The fibres form the comma tract of Max Schultze. They give off collaterals at different levels to the gray matter of the posterior horn, some of which decussate in the posterior commissure. They probably keep entering and leaving the white matter at different levels. At various levels they pass backwards to enter into the formation of the posterior internal triangle. At the first lumbar they leave this position to form the septo-marginal tract.

the column. Although these endogenous fibres can be distinctly seen owing to the complete atrophy of the exogenous systems, yet, as the chart shows there is a great outfall of these fibres in the whole of the dorsal and lumbo-sacral regions, the descending system being more affected than the ascending. Briefly, the degeneration corresponds with the advanced second stage or commencing third stage of ataxy, and the marked atrophy of roots in the upper dorsal and lumbo-sacral regions corresponds with the anæsthesia and analgesia observed during life, more marked on one side than the other.

The degeneration of roots and posterior columns in the eighth cervical and first dorsal, more marked on one side than the other, corresponds with the more marked sensory disturbance (observed during life) on the left than the right side.

The fact of the existence of the gastric crises may be associated with the marked degeneration of the roots in the mid dorsal region, and especially with the obvious affection of the gray matter in the fifth and sixth segments. It will be observed that there was no direct or crossed pyramidal degeneration, except possibly in the lowest lumbar and sacral regions, and this would accord with the fact that there was little chromophilous change in the Betts' cells of the cerebral cortex.

Brain.—Examination by cell and fibre methods. There is very little vascular change in the cortex as a rule, but typical plasma cells were found around the vessels in the orbital region; also excess of lymphocytes and endothelial cell proliferation; there is proliferation of the glia cells, but not many spider cells are seen. Sections of the parietal lobule, ascending frontal, ascending parietal, pre-frontal, orbital and angular gyri were made, stained by Nissl's method and examined. In all these regions the cells of the molecular layer of Cajal, the small and medium sized pyramids were found diminished in numbers, distorted in shape, not arranged as a rule in columns, their processes are broken off, and the bodies of the cells present various atrophic and degenerative changes of varying degrees of intensity, although some of the cells exhibit a fairly normal appearance. The cellular changes were more marked in the prefrontal and frontal regions than elsewhere. These changes appear to be of the nature of a primary cell atrophy. The Betts' cells in the ascending frontal are in groups, and some cells of a group appear fairly normal, while others show chromolytic changes, eccentric nucleus, and breaking off of the processes, indicative of an acute change.

Examination of the same regions by the Weigert method showed corresponding atrophy and partial disappearance of the tangential, supraradial and interradial fibres most marked in the regions where the cell atrophy was most apparent.

Case 32.—*Early ataxic general paralysis in a young married woman, the subject of syphilis (acquired before marriage, in all probability). Death in less than a month after admission from pneumonia. Left knee-jerk absent, right-sided fits, speech affection, right hemisphere weighed* 20 *grammes more than left.*

H. W., aged 26 years, admitted to Claybury, June 21, 1901.

History (from husband).—No family history of insanity, patient's brothers and sisters all strong and healthy. Married seven years ago at the age of 19, and before marriage she was an upholsterer by trade. Husband states that occasionally she has been drunk, but he is sure that her illness was not due to drink. She was an attractive-looking, good, and affectionate wife, and came from a very steady, good-living family. The husband attributes her illness to an accident she met with while with child; this child was still-born with difficult labour. Subsequently she had five or six miscarriages, but no children. For six months, or possibly longer, as she hid it from her husband, she suffered with a bad leg. Patient was the second wife of Mr. W., and in about September last he lost his daughter, aged 10, a child of his first wife, and the combined worry, husband thinks, constituted the exciting cause of the patient's illness. He denies that he ever had venereal disease, and I judged after the interview with him that his wife had been infected before having relations with him. Patient never complained of pains in her legs, except from the ulcer. As her illness progressed she had visual hallucinations of policemen coming into the house; she also had delusions that each person she met was a policeman, and that he was coming to lock her husband up. For the last eight or nine weeks husband has noticed that her speech has been affected. She had two or three fits before admission, the mouth being drawn to the left side, following the fit, but afterwards it came to the normal position.

Condition on admission: Mental.—Rambling and incoherent in her speech, believes that she has not been out of her house for five to seven years. *Physical.*—The right pupil larger than the left, both fixed; the right knee-jerk present, the left absent. She was suffering with palmar psoriasis and a syphilitic ulcer of the right leg, with the usual characteristic brown pigmentation

around. The teeth were worn down, otherwise her conditon was good, yet she was unable to walk. Her speech and expression were characteristic of general paralysis. The memory was very much impaired, and she was dull and sluggish in answering questions, and she had no knowledge of time or place. When questioned she jumps up as if to attention, but cannot maintain herself in the standing position.

Died July 7, 1901.

Post mortem.—Body well nourished, physique good, muscular system developed, *post-mortem* rigidity just commencing. On the right leg there is an old ulcer 1 in. by 2 ins., with pigmentation all round, the extent being as great as could be covered by the hand, typically syphilitic. The pupils were irregular, but equal, both being 5 mm. in diameter. There was no obvious thickening of the pia-arachnoid, but sub-pial hæmorrhages over the frontal region; it was adherent along the mesial surface of the frontal lobes. Weight of encephalon 1,120 grammes, appearance normal, with the exception of the hæmorrhage referred to. Weight of right hemisphere 490 grammes, weight of left 470 grammes, weight of cerebellum and pons 160 grammes. There is some apparent wasting of gray matter n the pre-frontal regions of both hemispheres. The ventricles were not granular or dilated. The lateral sacs of the fourth ventricle were slightly granular.

Right lung, weight 450 grammes, lower lobe in a state of commencing red hepatisation, commencing pneumonia of upper. Left lung, weight 650, lower lobe semi-solid, sinks in water, in a state of red hepatisation, commencing pneumonia of upper lobe.

Heart and valves all healthy, no atheroma aorta. Liver rather pale and fatty, spleen softer than normal, kidneys pale, otherwise normal. Stomach, pancreas, &c., normal, intestines normal. Os uteri dilated, tough and fibrous, cyst in left ovary the size of a pigeon's egg.

Cause of death, immediate.—Double pneumonia.

Other pathological conditions.—Very early general paralysis.

General summary.—Had she not contracted pneumonia a week or two after admission, she might have lived several years, as her general condition was excellent.

Case 33.—*Tabetic general paralysis in which the mental symptoms preceded the cord symptoms. Death within three months of admission. Characteristic brain and cord lesions.*

E. A., aged 32, married, policeman; late Army Service Corps, admitted to Hanwell, October 26, 1899, suffering from symptoms of general paralysis (tabetic form).

Family history (obtained from wife).—He is one of a family of eight, all alive and healthy, father and mother both healthy, no history of insanity or other neurosis, no history of phthisis.

Personal history.—Informant has been married to him for ten years; as far as she knows he was always bright, happy, and healthy as a young man; was a good husband and father; was in the army for ten years before marriage; they have had four children, eldest 8, youngest 2, one child died, aged $2\frac{3}{4}$ of pneumonia. Wife has had no miscarriages, and no history of syphilis can be obtained from her. She has seen him occasionally the worse for drink, but denies that he was habitually a heavy drinker. After the death of his child eighteen months ago, she first noticed that he became absent-minded, forgetful and melancholy, and he was obliged to leave the police force. His speech was first affected about two months ago; he has had no fits as far as she knows; his gait has become affected since his admission to the Infirmary three weeks ago.

He has a scar on glans penis, and two large scars in right groin, shotty glands in both groins; in spite of the above evidence, he denied having had venereal disease.

Physical condition.—Body somewhat emaciated, heart and lungs free from any symptoms of disease, tongue clean, tremulous, protruded slightly to the right, gait staggering, marked incoordination of muscles of locomotion and inability to walk without support. The unsteadiness is increased with the eyes closed and there is some difficulty apparent in touching the tip of his nose. The knee-jerks are absent, and there is some hypotonus of the hamstring muscles. The pupils are small, the right a little larger than the left and give no response to light; accommodation active. Speech most inarticulate and slurred, making it difficult to understand what he says. There is marked tremor of the lips and tongue. Slight drooping of both eyelids. He complains of rheumatic pains in his legs. No loss of sensation was observed on testing him by pricking and touch.

Mental state.—Confusion of ideas, impairment of memory, and mild delusions that he has money in the bank. He is restless and sleepless.

November 30.—Paresis rapidly progressing, has lost control over his sphincters, but asks to be changed and cleaned; he is rapidly emaciating.

December 11.—Commenced having epileptiform seizures, and he died on December 13 from acute pneumonia.

The brain showed the macroscopic signs of early general

paralysis ; the pia-arachnoid was slightly thickened and adherent over the fronto-central convolutions, erosions occurred on stripping, the gray matter was diminished, striæ indistinct. All the ventricles were somewhat dilated and granular. The spinal cord presented no naked eye change, but microscopical examination revealed early tabetic atrophy, as the subjoined microscopical examination shows :—

Fifth lumbar.—Slight diffuse sclerosis of posterior columns, except in the cornu commissural zone and the oval area. Slight diffuse sclerosis of crossed pyramidal tracts. Eighth dorsal.—Slight diffuse degeneration in posterior median column. Well-marked sclerosis of cornu radicular zone. Second dorsal.—Ditto Seventh cervical.—Slight degeneration of posterior median, better marked degeneration of band just outside this, coming, doubtless, from eighth cervical, first and second dorsal-fibres. At the base this spreads out to reach the posterior horn.

In no part of the cord were the endogenous fibres of the posterior columns obviously degenerated. The posterior roots show atrophy and denudation of fibres, especially in those regions where the posterior column degeneration is marked. The vessels and membranes show little or no change, and the cord presents little change in size or shape, for there is comparatively little wasting.

Case 34.—*Ataxy and slight early seizures and symptoms of general paralysis, abeyance of ataxic symptoms, pronounced mental symptoms following seizures.*

F. J., compositor, aged 49. Admitted to Colney Hatch. My attention was called to him by Dr. Seward on account of his ataxic walk. He gave a history of syphilis, scar on penis, and from his own account he had evidently lived a very rackety life. There was no history of insanity in the family. When I first saw him he was not much affected mentally, but he was ataxic ; he had pains in the legs, unsteadiness in his gait, which was increased on closing his eyes. The pupils were equal, contracted to 3 mm. and did not respond to light, but sluggishly to accommodation. The knee-jerks were absent, and there was hypotonus in the limbs. The speech was hesitant and somewhat syllabic, and the tongue tremulous. Six months later when I saw him he was a most pronounced general paralytic ; he had had seizures, and the speech was characteristic of advanced general paralysis. He had delusions of wealth, grandeur, and strength, and a markedly

exalted expression (*vide* photo). His walk was now a shuffle, but he could stand without any swaying with his eyes shut. The knee-jerks are still absent, but there is no hypotonus in the limbs. He subsequently died. The brain presented all the characteristic appearances of general paralysis. The spinal cord was forwarded to me for examination with the following results :—

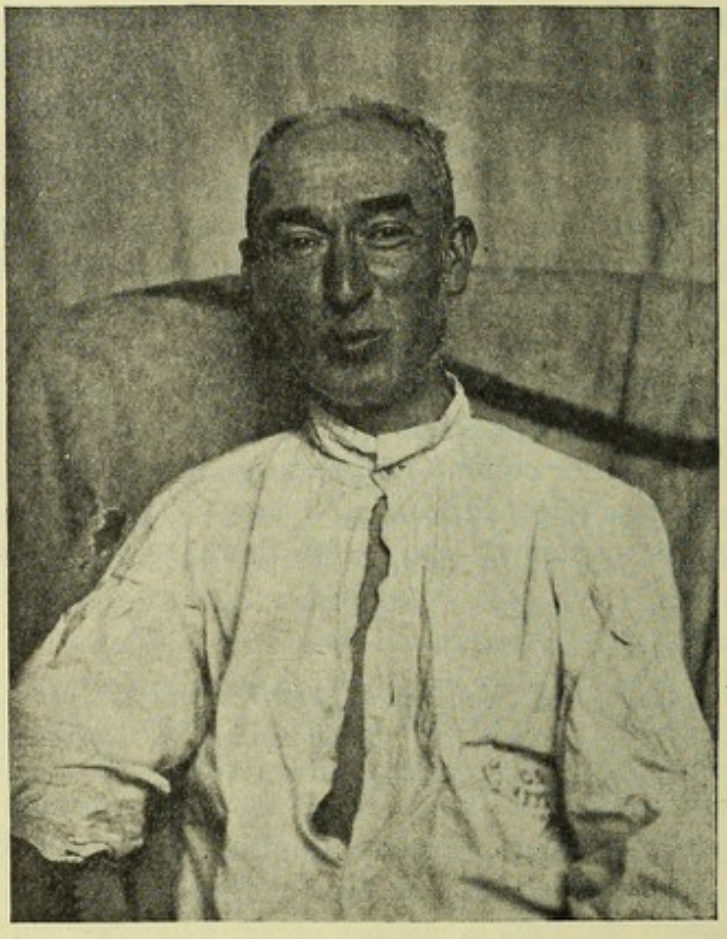


FIG. 25.

The photograph illustrates a condition of marked exaltation and grandiose delirium ; at the time this was taken the patient was telling me that his brain was geared up to 990,000.

Microscopical examination.—The sclerosis, although most obvious in the exogenous systems of the posterior columns, is not limited thereto, as there is a very definite sclerosis in the comma tract, but the cornu commisural zone of Westphal in the lower dorsal and lumbo-sacral regions is intact. There is diffuse sclerosis of Goll's column in the cervical region and a well-marked

band of sclerosis corresponding to roots supplying the upper limb. There is well-marked atrophy and denudation of fibres in the middle third of the postero-external column corresponding to Charcot's root zone in the dorsal and lumbo-sacral regions, with atrophy and disappearance of many of the fibres of the plexus around the cells of Clarke's column. This atrophy is most apparent where Clarke's column is most developed —viz., in the lower dorsal and the upper lumbar regions.

The median oval area of Flechsig, and Gombault and Phillipe's posterior triangle appear to be only slightly denuded of fibres. There is some acute degeneration of the crossed pyramidal tracts on both sides throughout the cord, and a little degeneration in the direct tracts (*vide* chart).

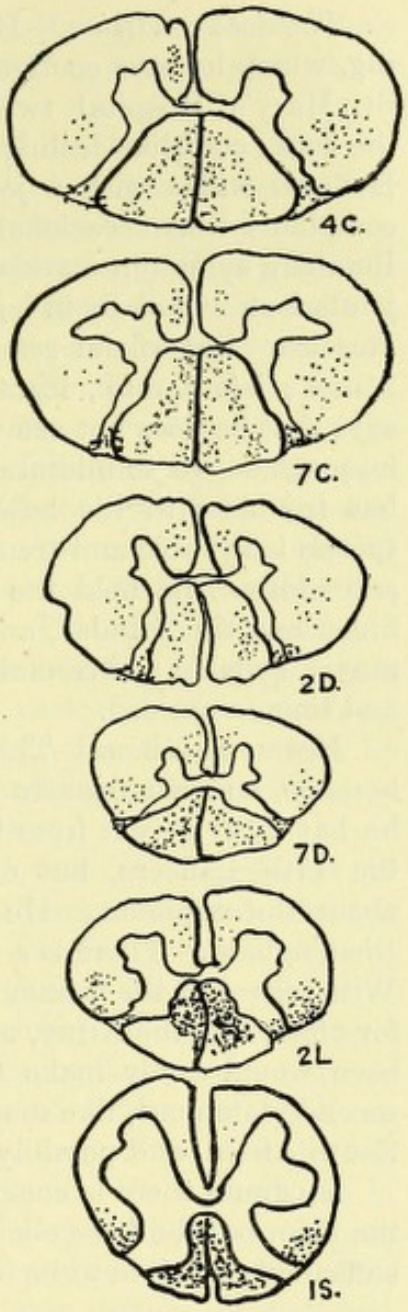


FIG. 26.

Case 35.—*Tabes of two or three years' duration, very advanced in legs and arms. Gastric crises. Bladder and bowel troubles. Admitted to asylum suffering with mania, exalted notions, and intellectual confusion.*

N. A., admitted to Colney Hatch May, 1901, Russian Jew, aged 33, a tailor by occupation, has to do a great deal of walking, and always stands at his work as a cutter. He has been fifteen years in England, married nine years. After marriage, wife had four miscarriages, then three children died in early infancy, finally two living. At 15 years of age the patient contracted a chancre, which was treated for two years, suffered with sore throat, rash, and falling out of hair. There is no scar visible; glands in the groin are enlarged. He is a temperate man, smokes; there is no history of insanity in the family.

Physical condition.—He has a difficulty in standing and walking, which he says commenced two years ago. He was treated at St. Mary's Hospital twelve months ago for shooting pains in the legs, cord-like feeling round the waist, and loss of memory. He now walks with a jerky wide base, heels down first. He complains that occasionally his legs have given way under him. Romberg symptom marked, knee-jerks absent, sense of position of joints lost, especially in legs, very marked hypotonus of hamstring muscles. Superficial reflexes exaggerated, pupils unequal, right 2½mm., left 1½mm., inactive to light, active to accommodation, says that he does not see well with the left eye. Complains of a frequent desire to micturate, and difficulty in passing water. He has trouble with his bowels and bad attacks of vomiting. His speech is syllabic and tremulous. Being asked to write his name and address, he held the pencil between the side of the middle finger and tip of index, and the writing, like the speech, was very ataxic, syllabic and tremulous. There was slight tremor of the lips and tongue noticed.

Mental condition.—There is some mental confusion and incoherence, and a moderate amount of exaltation. He thinks that he has £250 a year from the War Office, that he is a General in the 17th Lancers, but on questioning him, he knows nothing about the regiment. He is the owner of a diamond factory in Birmingham. There is a sub-stratum of truth in these delusions. With regard to the former statement, he probably makes uniforms for officers in the Army, and a good cutter, such as he may have been, would easily make £250 per annum. With regard to the second statement, like many other of these aliens, he has been in South Africa, and possibly in the illicit diamond trade.

No doubt there is considerable disturbance in the sensibility of the skin, but the facts elicited on this occasion were not considered sufficiently reliable to be included in the notes.

Case 36.—*Tabo-paralysis, commencing with a fit, followed by transitory aphasia. Later typical signs and symptoms of ataxy, melancholia, attempted suicide, progressive degeneration of brain and cord, with delusions and illusions, death, with typical naked eye signs in the brain of general paralysis.*

A. H., aged 38, admitted to Hanwell, July 31, 1896, for melancholia and attempted suicide, which he nearly succeeded in accomplishing by cutting his throat, the scar of which is evident. Occupation, fireman on an engine, standing all the time, exposed

to weather and the heat of the furnace. He attributes his ataxy to his occupation, and he is very depressed in consequence of his being unable to continue his work.

Family history.—Maternal uncle died in an asylum.

Personal history (from the wife).—She has known the patient for eleven years. During this time he has always been ailing. For the past five years he has been much worse. Five years ago he had a seizure of some kind, and lost power of speech for two days. Since that time his speech has become much worse. In 1892 he found a difficulty in walking in the dark, and he had a velvet-like feeling on the soles of the feet. Twelve months ago he complained of a girdle sensation, followed by what were evidently rectal crises. Later on he suffered with loss of sexual power. His mind became affected after a severe fit which he had fourteen months ago. On the 1st of June last he had another fit, remaining unconscious for one hour. The medical certificate states that he is simple and childish, laughing insanely when spoken to. He had collected a lot of useless buttons, pebbles, bits of clay pipe, and attempted to explain the uses to which he would put them. His statements are rambling and contradictory. Twelve months ago he attempted suicide.

Present state (August, 1896).—He has an expression of depression, but I could discover no delusions or mental affection beyond this extreme depression, due to his being in an asylum, and unable to maintain his wife and children. His knowledge of time and place are good, likewise his memory for recent and past events. There is no tremor in the tongue, or face muscles, nor is the speech obviously affected. There is no ocular paralysis, no signs of syphilis on the body, and no history of the disease, but the possibility cannot be absolutely excluded. He is temperate in habits. He has a *marked ataxic gait*, Argyll-Robertson pupils, absence of knee-jerks, loss of sense of position in lower limbs, not in upper; complete loss of power over rectum, partial over bladder, cutaneous anæsthesia could not be determined in a reliable manner, owing to his refusal to answer.

November, 1897.—Patient suffered with severe epileptiform attacks, followed by delusions of persecution. People are putting dung in his food, and are playing upon him with electricity ; his gait is not so ataxic.

August, 1898.—He is still suffering with delusions of poisoning, accompanied by great mental depression. He had been suffering with dysenteric diarrhœa, and he considers it was due to the medicines and food.

April 27, 1899.—The notes state that he has become paralysed on the left side of the body; he is still suffering with delusions of poisoning, and people playing upon him with electricity, and he is more demented.

July 31, 1899.—Patient is certified as dying from dementia and paralysis. The *post-mortem* notes state that the brain weighed 46 ozs.; the convolutions on the right side were the seat of extensive atrophy and white softening in the ascending frontal, first temporal and angular gyri; wasting of the left leg is also noted.

Case 37.—*Tabo-paralysis. Grandiose delusions, acute mania, macropsy, slight ataxy, pains attributed to electricity, auditory and visual hallucinations. Death eight months after onset of mental symptoms.*

N. E. E., aged 46, admitted to Hanwell, June 28, 1901; occupation woodcarver and turner. His work necessitated standing all day.

Personal history (from wife).—He has been worried lately a good deal by the foreman on account of his inability to perform the work satisfactorily. He has been married twice, but has no children. His first wife had one miscarriage. He has been married to his second wife sixteen years. Patient's father died raving mad, was very intemperate, probably *mania à potu*, as the delirium was only a few days before death. An aunt was very eccentric, no history of phthisis. Patient when very young had fits between the ages of 4 and 6, but none since. He has been a steady, temperate man, thoroughly moral and very clever and industrious. The wife says that two doctors pronounced the opinion that the patient had syphilis when he was a young man, for which he was treated for some time. The patient himself, when questioned about this, said it was what is called a soft sore; it was only treated locally.

History of present illness (from wife).—Eight or nine years ago he suffered with dyspepsia and dilatation of the stomach. He has always been an excitable man, and his health has broken down latterly. He has complained of his eyesight, and he has had sharp shooting pains in the legs. The mental symptoms started about six months ago with extravagant ideas.

Physical condition.—Gait not ataxic, but walks with a slightly wider base than usual, Romberg symptom is absent, muscles well developed and strong, knee-jerks absent, superficial reflexes exaggerated, some hypotonus of hamstrings, legs can be raised

nearly to right angles with the body, no loss of joint sensation, no loss of sense of position. Pupils 2 mm., equal, irregular outline, inactive to light and pain, active to accommodation. When looking straight forward, the pupils dilate upon his entering into conversation. He tells me he has the *sensation of everything appearing very large.* This statement he volunteered. Colour vision is good, he can read small print, and the fields are not limited.

Expression anxious and depressed, but when conversing, his face becomes very animated and emotional, and there is tremor in the lips. The tongue also is tremulous, the speech, however, is but very slightly affected. There is no history of fits before admission or since. He has had no feeling of constriction round the waist, and there is no difficulty with the water or the bowels. No scar or enlarged glands can be detected, but there are numerous small-pock-like scars on the forehead and a few on the legs. I could detect no cutaneous anæsthesia. His handwriting is good, and like his speech flowing and not tremulous, but it does show slight cutting up of syllables, (?) pathological.

Mental condition.—When brought to the asylum he was singing the Marseillaise with patriotic fervour. He is now full of grandiose delusions, some of which have a basis of truth, *e.g.*, he says that he is a great singer. He illustrated this by singing several songs in French with considerable expression, excellent articulation, and with a fine resonant voice. He then burst forth into the Marseillaise, singing it with extraordinary stirring patriotic fervour, suiting the action to the words. He was very pleased with his own performance, and spoke of kings, diamonds, and wealth. During his conversation, the muscles of expression associated with hilarity were especially brought into play, and the pupils dilated, but on ceasing, the same wearied lack of expression occurred.

August 20.—Patient in a side room, pale and emaciated, continually talking to imaginary persons, making lunges at the wall, or jerking his hands in the air as if throwing off some imaginary objects on his body. For the last few days he has employed himself rubbing his skin, especially of the knees and feet, with his shirt or bedclothes, muttering "electricity." He has thus caused several sores on his body. These symptoms indicate auditory and visual hallucinations and lightning pains in the legs.

The patient became very emaciated and died September, 1902. The brain showed characteristic signs of general paralysis, and

the spinal cord exhibited early tabic degeneration of the posterior columns in the lumbo-sacral region, and slight sclerosis of Goll's column in the dorsal and cervical regions, after hardening in Muller's fluid.

Case 38.—*Tabetic general paralysis, onset of tabetic and mental symptoms apparently simultaneously, arrest of tabetic condition, but slowly progressive dementia, symmetrical perforating ulcers of feet, knee-jerk absent on right side, present on left, speech for a long time only slightly affected, moderate exaltation and incoherence, knowledge of time and place not markedly affected.*

W. D., aged 38, butler, married, with six children, admitted to Colney Hatch, November, 1897; history of six months illness, and certified as suffering from early tabetic general paralysis, history of syphilis at the age of 16, pigmented scar of old gumma on leg.

Physical condition.—Tall powerful man, walks with rather a wide base, heels down first, but no incoordination or jerkiness in his movements, can stand with his eyes shut, knee-jerk absent on the right side, present on the left. He can touch the tip of his nose with the forefinger of the left hand, but not accurately with the forefinger of the right.

Speech, no slurring of syllables, but slight hesitancy, can utter test words and sentences fairly well, without elision or slurring of syllables. There is no tremor of lips, and very slight of tongue.

Mental condition.—Mild dementia and confusion, but no marked loss of knowledge of time or place. He has delusions, but there is a substratum of truth, *e.g.*, he says he knows all languages. Having been in the service of the French Ambassador as butler, he has probably heard many languages spoken, and doubtless, occasionally picked up a few words; certainly he knows a few sentences of French, German, and Italian, but he fails to understand simple questions put in French or German, although he will make an attempt to reply in the language by which he has been addressed. A few months after he had been in the asylum he developed two symmetrical perforating ulcers of the feet. The ataxy did not increase, but the dementia slowly progressed. He had no fits.

In October, 1899, he told me his age was 27, that he had a daughter who was married. There is a little more speech

affection, and the attendant says he is very restless at night, getting up continually to make his bed and brush out the flies which he imagines are walking over him (?) formication. He appears to feel pricking and touch in all parts of his body, but it is difficult to be certain on account of his mental condition.

November 14, 1900.—The left foot and leg are much swollen owing to an infective cellulitis, the knee-jerk on this side is still obtainable, but is absent on the right. The speech is more hesitant and slurred, knowledge of time and place more defective, and he now has grandiose delusions of wealth. He states that he has lots of money in the bank, and he will present each of us with a gold watch.

June 1, 1901.—Dementia more marked, has a delusion that he is the Prince of Wales, expression exalted. Nutrition fairly good, appetite ravenous, knee-jerk cannot now be obtained on either side. Pupils each measure 3½mm., irregular in outline, inactive to light, but react to accommodation. For a long time past he has persisted in getting up in the night to make his bed, he continually brushes the sheets. When the attendant asked why he did this, he said there were flies in his bed walking over him; very possibly this is a delusion based upon formication, owing to the cord affection.

Case 39.—*Advanced tabes of at least four years' standing. Delusions of persecution and auditory hallucinations then developed with mental excitement. Slight organic changes in the brain, especially of the frontal lobe, most advanced spinal sclerosis in posterior column.*

L. W. K. Admitted February 1, 1900. Stockbroker's accountant, aged 50. Single.

Previous history (from brother on July 13, 1900).—Maternal grandfather drank heavily, and a distant relative has been in Earlswood for more than thirty years. He had syphilis between twenty-five and thirty years ago. Abstemious for many years, but previously drank. He has had a considerable amount of mental worry. During the past two years at least, and probably for three or even four, he had walked very badly, raising his feet high from the ground; the pupils of his eyes were like pinpoints. His present mental condition began quite suddenly just after Christmas, 1899. Shortly before Christmas he seemed very jovial and behaved in an artificial manner, and a week after Christmas he began to ramble in his conversation. He said that people

were posting notices on walls about him. He told his brother not to go to his office, as there was a conspiracy against him. He wandered from home for the whole of one day, but returned of his own accord. He slept badly during the week referred to, he was then admitted into Hammersmith Infirmary, and a week later was transferred to Claybury.

Facts stated in certificates.—Patient is excited, hears voices, says there is a conspiracy against him, that his brother has stolen his money, and that he knows himself that he is suffering from hallucinations. He jumps up in bed at night and calls out. Brought to the asylum in a straight jacket.

Present state—Physical.—Height 5 feet 7 inches, weight 9 stone 4 lbs. Two broken ribs on left side. Tongue coated, appetite good. Bowels irregular, urine and viscera normal, pupils irregular, contracted, and do not react. Unable to stand with eyes closed, gait tabetic, knee-jerks absent. *Mental.*—Dazed, lost, confused, and depressed, very restless, and constantly wandering about, short-tempered, impulsive, violent, and given to striking out at the attendant. He hears imaginary voices accusing him "of stealing and owing money," heard "his brother groaning in the next room," sleeps badly, but takes his food well, and is clean in his habits.

Is suffering with mania and from tabes, is emotional, and his statements are contradictory and incoherent. He believes there has been, and there is a conspiracy against him for stealing a young lady's jewels, and assures me that it is false. His memory is impaired. He believes that some one is pointed out to him as his brother who is not, and he has aural hallucinations. He is full of nonsense about a certain young lady, and the wrong he has done his brother. He feels that his food has been tampered with for years, because he has seduced his mother's servant and given her syphilis, and says that her husband has been following him to take his life. He is in poor health and fair nutrition. He cannot stand with his feet together blindfolded. His gait is tabetic, his pupils are contracted, pinpoint, and inactive. He admits syphilis twenty-five years ago, he complains of shooting, neuralgic pains in his legs for some years, although nothing, he states, is to be seen.

February 21, 1900.—Impulsive, quarrelsome, and violent, but does not now appear to be troubled with auditory hallucinations.

April 30, 1900.—He is considered to be a general paralytic. Signs of progressive dementia.

August 1, 1900.—He is now constantly wet, and very ataxic.

October 23, 1900.—Stamps with his heels in walking, and throws his legs about somewhat, tongue very tremulous, speech rapid and slurred and without "r's." He is failing very rapidly, and is very wet and dirty in his habits. The knee-jerks are absent, there is hypotonus to the extent of 20° beyond the vertical. When relaxed, the right pupil is $1\frac{2}{3}$ mm., and the left $1\frac{2}{3}$ mm., the pupils rapidly accommodate, the right to 1 mm., and the left to 1 mm. Both pupils are irregular and inactive to light.

October 30, 1900.—Rapidly became feebler, and died this morning at 9.50 a.m.

Abstract of notes of post mortem.—Poorly nourished, bedsores on both buttocks and over sacrum, doubtful scar on glans penis. *Head.*—Skull cap dense, dura mater natural at the vertex, thin brown film above the tentorium and in the anterior and middle fossæ, and most marked on the left side. Slight excess of subdural fluid, pia-arachnoid somewhat thickened, and strips rather more readily than natural. No adhesions in the mid line of the pre-frontal region, but there is considerable granularity of the pia in this situation. The pia here is also adherent to the subjacent cortex, which is congested. The basal vessels are natural. There is no definite change in the optic nerves; the left olfactory tract appears to be smaller than the right, and both seem to be smaller than natural. Brain weighs 1,395 grams, natural, except for some fronto-parietal wasting. Each hemisphere weighs 600 grams, cerebellum and pons weigh 175 grams; the brain having been weighed without draining, this only leaves 20 grams of fluid. The hemispheres are normally convoluted. The cortex is dark and congested; the rest of the brain is not congested, but the whole is somewhat wet, without the condition being that of definite œdema. The lateral ventricles are slightly dilated, and contain a few doubtful granules. The fourth ventricle throughout its extent appears to be covered with small granulations. After the hemispheres had been in formol for two days, and the ependyma had become hardened, all the ventricles were obviously granular. Cartilages natural, ribs brittle. On each side of the thorax there was evidence of old-standing fracture of five or six of the lower ribs in front of the mid axillary line. No pleural adhesions. Both lungs broncho-pneumonic and œdematous. Throughout the aorta was a large amount of atheroma (first and second stages); there were no calcareous plates, but numerous pearly-white patches of fibrosis. Ureters inflamed throughout their course, acute cystitis, testicles natural. The brain, after

having been hardened in Muller's fluid, showed some thickening of the pia arachnoid over the motor area, particularly the upper part, and Broca. The above-named convolutions were shrunken.

Microscopical examination of the brain.—Sections stained by Nissl method showed acute changes in the small, medium, and large pyramids, and overgrowth of glia cells. There was some cell proliferation of the perivascular lymphatics in Broca's convolution. The left ascending frontal and parietal showed glia cell proliferation, especially in the deeper layers of the cortex, acute degenerative changes in the nerve-cells, and displacement of Meynert's columns. In the posterior part of the first left temporal there was very little change observed; the same applies to the calcarine. Orbital mesial surface.—The vessels showed abundance of plasma cells in the lymphatic sheaths. The pyramidal cells had lost their normal shape, and many of them were atrophied, but there was no marked glia cell proliferation in this region.

Marchi method.—A slight amount of recent degeneration was observed in the white matter. In the ascending frontal and parietal there was considerable recent degeneration of radiating fibres, more obvious than usually met with in general paralysis cases; fine black dots could be traced into the second layer, but, probably, these fibres do not come from the pyramids of the third layer. Nothing noteworthy is to be seen in the first temporal or calcarine; only a few degenerated fibres can be seen, but they are practically inconsiderable in number, compared with those of the fronto-parietal region.

Marchi-Pal.—Broca's convolution.—The tangential and superradial fibres are absent, and the interradial diminished in number. Base of first frontal, ascending frontal, ascending parietal.—In the first-named convolution the tangential fibres are absent, in the other two they are greatly diminished, and in places absent altogether. The super and interradial fibres are less affected than the tangential, but they are diminished in number; there is no thickening of membranes and but little congestion of vessels. First temporal convolution—there is a marked diminution of tangential fibres, less of superradial and interradial. There is no apparent thickening of membranes or vascular change noticeable. The same remarks apply to the occipital lobe in the region of the calcarine fissure, except that the vessels are passively congested (due to position). In the orbital lobe the tangential and superradial fibres are entirely gone, and the interradial greatly diminished in number, the cells stained with osmic acid as if they

were undergoing fatty and pigmentary degeneration. The spinal lesions are shown in the accompanying diagram which explains itself. It may, however, be remarked that each segment of the cord was examined and drawn by means of an Edinger projection apparatus, and it will be observed that there is no appreciable flattening of the posterior surface of the cord, although there is very obvious shrinkage in the transverse diameter of the posterior column. This also is shown strikingly in the photomicrograph. From the second to the eighth dorsal there is almost complete absence of fibres in the posterior roots at their attachment to the

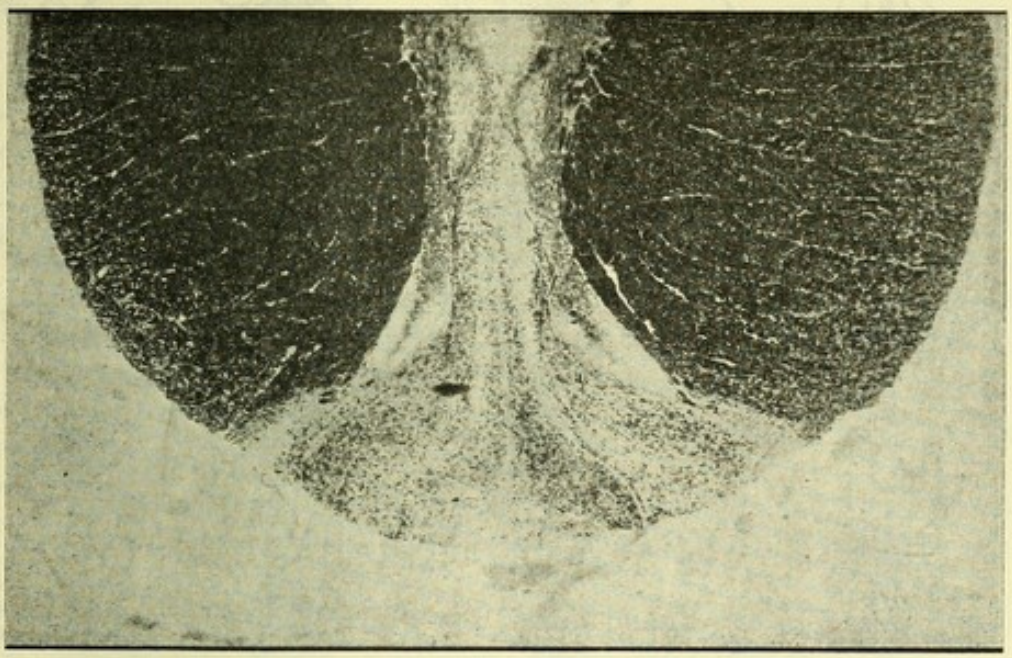


FIG. 27.

Section of spinal cord at level of eighth dorsal segment. There is no flattening of the posterior column; it is even more convex than normal. There is, however, very marked shrinking and distortion of the gray matter and of the posterior column, so that the columns of Clarke are almost in apposition. The fibre plexus around the cells is completely atrophied, but encircling the columns are two sets of fibres, one external, proceeding from the gray matter of the posterior horn, the other internal, continuous with the fibres of the postero-external zone.

Magnification 16.

cord, and in the same region a very complete degenerative atrophy of the exogenous fibres in the same regions of the cord. The degeneration below this eighth segment is not so marked but still very extensive. The endogenous tracts are atrophied but to a much less degree. Black indicates complete absence of fibres, dots partial absence. It will be observed that there is scattered crossed pyramidal degeneration which, however, cannot be traced above

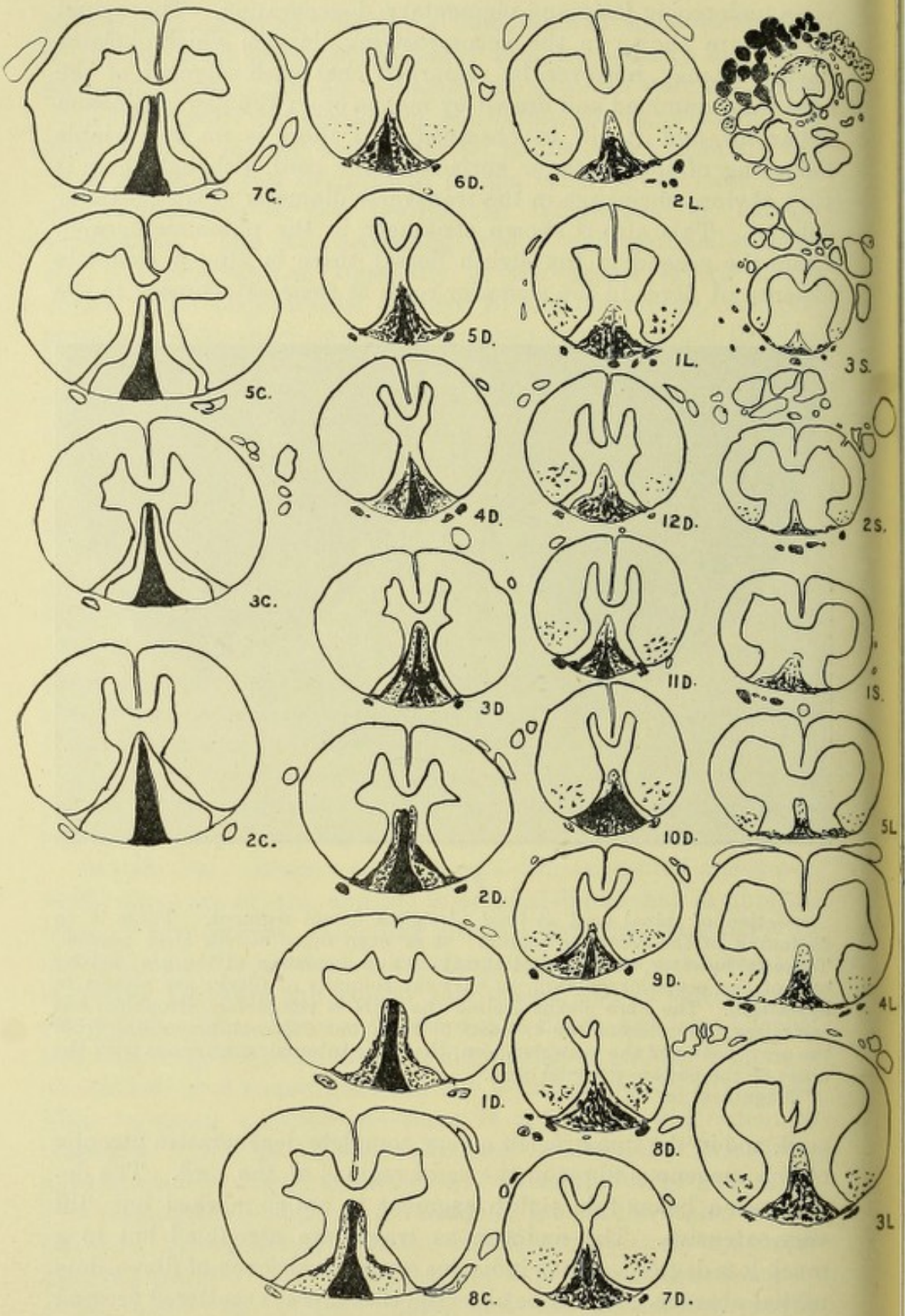


FIG. 28.

Note degeneration in crossed pyramidal tracts, which ceases in mid-dorsal region.

the mid-dorsal region. The posterior spinal ganglia showed only some pigmentary atrophy of the cells. The fibres distal to the ganglia were normal, whereas the central proximal fibres were completely or nearly completely devoid of myelin or had entirely disappeared. It was noticed that, whereas the myelin could be seen on some of the attenuated fibres, as they proceed centrally from the ganglion, after a short distance it seemed to disappear. The atrophy of the fine plexus about the cells of Clarke's column was very extensive, indicating marked affection of the cerebellar path. Lissauer's zone was also pretty extensively atrophied in the lumbo-sacral region; it was, however, variable on the two sides and at different levels, corresponding pretty closely with the degree of atrophy of fibres in attached roots.

Nerves.—Right posterior tibial. Longitudinal and transverse sections show some degenerated fibres, but the degeneration appears to be not the Wallerian degeneration, but a swelling and opacity of the myelin in many of the fibres by which it stains imperfectly. Some of the fibres show merely empty primitive sheaths, and in longitudinal section these fibres are seen at intervals to exhibit coagulated balls of myelin or smaller myelin droplets. The anterior tibial nerve shows this change even more distinctly.

Transverse sections of the sciatic nerve show paucity of fibres in many of the bundles, but whether there is a real atrophy or not it is difficult to decide.

Skin of great toe—very few small bundles of nerve-fibres are seen, certainly less than normal. Examined with a high power one sees not more than one-third of the fibres are stained blue.

Degeneration of the peripheral nerves, especially of the remote parts does exist, but it is inconsiderable as compared with the posterior roots.

Optic nerves—transverse and longitudinal sections show by Marchi method many recent degenerated fibres scattered over the whole field, but more marked at the periphery, where also could be seen many bundles showing advanced atrophic degeneration.

Case 40.—*Ataxy with slight mental symptoms, altered respiration from failure of synergic action of muscles of abdomen, complete destruction of all the fibres of the posterior roots in the lower dorsal region of the cord.*

F. S., aged 61, married, chairmaker; has had seventeen children, fourteen living. He had syphilis at the age of 25. He

was treated for it medicinally for a long time. His symptoms began with lightning pains, some years ago, exact time not known. He had been a considerable time in the infirmary, and was admitted to Colney Hatch, May 28, 1896, for mania.

Present condition.—No discoverable mental symptoms, except feeble-mindedness, he is markedly ataxic, muscles greatly wasted, has girdle sensation, lightning pains, knee-jerks absent, plantar reflexes absent, abdominal reflexes greatly diminished on left, absent on right side. Argyll-Robertson pupils. There is loss of muscular power in the legs, and marked hypotonus, but his arms still possess considerable strength. There

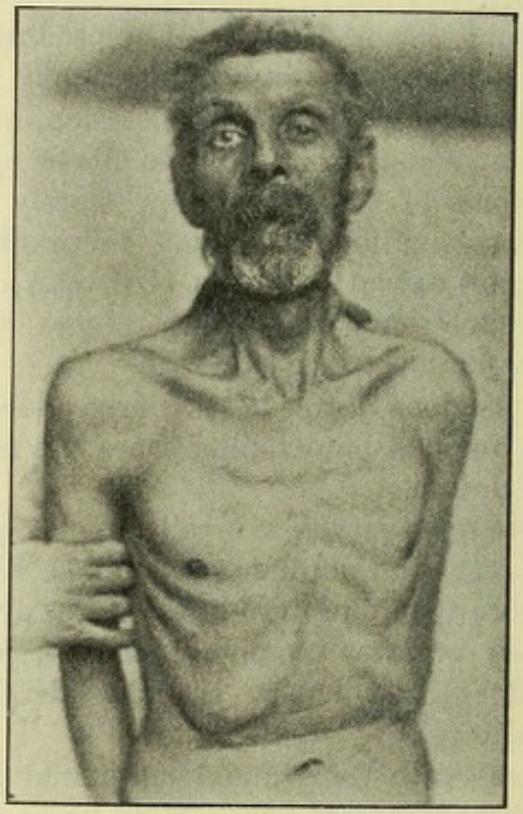


Fig. 29.

is marked incoordination of the lower limbs. With his fingers he can readily touch the tip of his nose, when his eyes are shut. The belly is retracted, and there is complete atrophy of the left, and partial atrophy of the right quadratus lumborum and oblique muscles. Upon taking a deep respiration the lower ribs are drawn inwards, and a groove is formed (*vide* photo.). There

is also great atrophy and weakness of the muscles of the lower extremities, they react to faradism, but require a stronger current than usual. There is no absolute loss of sensation anywhere in the legs, but it is much delayed and blunted, and there is numbness of the soles of the feet. The trunk was not tested; there is also complete absence of sexual power, and loss of control of bladder and rectum. There are symptoms pointing to rectal crises, but no gastric, larnygeal or other crises.

July 1, 1896.—Condition of patient much the same, but there has been gradual loss of muscular power, and he is now bed-ridden. Left abdominal reflex is now quite absent. (F. W. M.)

October 14, 1896.—Patient died to-day of exhaustion.

Autopsy.—Fifteen hours after death. Body extremely wasted, and there is a large bed sore over the sacrum. The viscera presented no signs of disease except the bladder, the mucous membrane of which was thickened and inflamed. Naked eye sclerosis was visible in the posterior columns of the spinal cord.

Microscopical examination (by Dr. Hamilton Wright) — *Brain.*—Some pia-arachnoid thickening in frontal and central regions, with chronic atrophy of superficial fibres belonging to tangential and supraradial systems. *Spinal cord.—Cervical enlargement.*—The postero-median columns are denuded of fibres, and markedly sclerosed, except at their extreme apices, in the position of the endogenous cornu commisural zone. The inner halves and almost the whole of the median parts of the postero-external columns, show marked sclerosis, with a very few fibres only left. A few fibres have disappeared from the root zones, and they are slightly sclerosed. Lissauer's tract on both sides is partially atrophied. There is a partial atrophy of the roots of this region, external to the cord. Many of the fibres still *in situ* are undergoing degeneration, and there is some fibrous tissue substitution. The right lateral column presents a marked degeneration, confined in the main to the anterior extremity of the direct cerebellar tract. The whole of this tract, however, is the seat of a diffuse fibre atrophy and sclerosis. On both sides there is a slight degeneration of fibres and sclerosis of the crossed pyramidal tracts. The Weigert-Pal method, which was employed for this investigation, revealed no degeneration in any part of the gray matter. Many anterior cornual cells are shrunken and much pigmented. There is a slight rarefaction in the posterior horns, probably the result of the few posterior root-fibres which course through them.

Thoracic region.—At this level there is a marked atrophy of fibres in the postero-median columns. A few only are left, and these are in a state of chronic atrophy. The postero-external columns, except in the most anterior parts, and along the median margin of gray matter, exhibit marked sclerosis. The fibres of Lissauer's tract have wholly disappeared ; all these parts are the seat of a marked sclerosis. A few fibres are left in the position of the cornu-commissural zone. The comma tracts are almost totally atrophied. The extra-cordal portion of the posterior roots contain no sign of nerve-fibres, but only a scattered *débris*. On the right side of the cord the crossed pyramidal tracts

FIG. 30.

Section of spinal cord at level of lowest dorsal region. There is complete atrophy of Lissauer's tract extending into the region of the direct cerebellar tract, each side of which is also atrophied. The entering root-fibres have completely disappeared. The fibres in the posterior column are mainly continuous with the two bands of fibre which encircle Clarke's column, and are in all probability endogenous. The fibre plexus around the cells of Clarke's column is completely atrophied. The central canal is dilated.

show an atrophy of quite one-third of their fibres, and are deeply sclerosed. There is only a slight atrophy and sclerosis in this tract on the opposite side. Both posterior horns are greatly wasted. In the right horn no proper tissue is to be observed dorsal to its neck. The opposite horn contains here and there a sign of proper tissue posterior to Clarke's column. Clarke's columns are denuded of fibres of all kinds. The right column of Clarke contains no evidence of nerve-cells, except in the lower segments, and even here they are greatly shrunken and deeply pigmented. A few only are to be observed in the opposite

column, all greatly shrunken and pigmented. The posterior gray commissure contains only a few vestiges of fibres. The gray matter of the anterior horns is shrunken and rarefied, its cells are deeply pigmented and exceedingly few in number. The anterior roots on the left side are almost totally atrophied. On the opposite side a few fibres are still to be observed. *Lumbo-sacral region.*—The only fibres left in the posterior columns of this division of the cord are in the position of the cornu-commissural zone, along the median margin of the left posterior horn and in the left root zone. Even here they are few, greatly attenuated, or irregularly swollen. All other parts are intensely sclerosed. There is no sign of the median triangle of Flechsig, or the sacral fibres of Gombault and Phillippe. This is an important point in view of the sphincter paralysis. The posterior roots on the right side are totally atrophied. A few extremely wasted are still present on the left side. In the lateral columns of this level the atrophy of fibres, and the consequent sclerosis is apparently not so great as in the thoracic region. It appears to be equal on the two sides. The posterior horns contain only a few fragments of fibres in the substantia gelatinosa. The cells appear to have totally atrophied. The necks of the posterior horns are greatly rarefied and sclerosed. Only a few remains of fibres are present in the posterior gray commissure, and none of these appear to bend backwards along the posterior median septum. The anterior cornual cells are shrunken and are deeply pigmented; it is difficult to decide whether any have totally wasted and disappeared, but it is not unlikely in view of the state of the anterior roots. On both sides the root-fibres have in great part disappeared, and the majority of fibres that remain are attenuated. The piamater is thickened, and the superficial layer of neuroglia beneath it appears to be augmented throughout the cord, especially where the sclerosis is marked. There is a great congestion of the pial, and deeper vessels; thickening of their coats has occurred in the pia, and in those parts of the cord where atrophy of fibres has been followed by sclerosis. *Posterior spinal ganglia* in the cervical region were not examined. In the thoracic and lumbo-sacral levels there is considerable degeneration of the cells. The most noticeable feature is a general pigmentation; in many instances no vestige of the cell is to be seen, and the capsule is full of dark brown pigment. Many cells have stained purplish blue by the Weigert-Pal method, indicating fatty degeneration. A few cells are reduced to a granular debris, in the centre of which is a turgid granular nucleolus, the nucleus

having entirely disappeared. The capsules are thickened, and the general interstitial overgrowth is marked. The vessels are congested, and their walls slightly thickened.

Case 41.—*Tabes, probably of some standing, homicidal and suicidal mania, grandiose delusions. Ataxia, mental and physical improvement, obvious ataxy disappears, attack of suicidal mania, left-sided convulsions, tremor of face muscles, but little speech affection. Death from intercurrent disease two years after admission. Naked eye and microscopical examination of brain and spinal cord exhibits first stage of ataxic lesion of cord and roots, heterotopia of central canal in lumbo-sacral region. Changes in cortex cerebri of general paralysis.*

H. J. B., telegraphist, aged 41, admitted to asylum, Cane Hill, December 28, 1897. Died December 14, 1899. Medical certificate states that he had threatened to shoot his wife and then himself. Before admission to the Infirmary he had been excited and emotional. He had suffered with unbearable pains, he had been the subject of grandiose delusions, pawned nearly everything for drink, stolen billiard balls, brushes and other articles.

History.—First attack. Duration of mental symptoms two months; first symptom noticed, sleeplessness and loss of flesh—has locomotor ataxy which began nine months ago; supposed cause, alcohol; married ten years, two children. No phthisis, insanity, or alcohol in family history. States himself that he had a soft sore for which he was treated with medicine for two months.

Physical condition.—Nutrition good. Tongue tremulous. Pupils unequal, Argyll-Robertson. Deep reflexes absent. Plantar present, marked. Gait, walks with a wide base. Romberg sign. Complains of pains, no visceral disturbances. No evidence of organic disease of abdominal or thoracic organs.

Mental condition.—Talkative, rambling, irrational. He has exalted notions of his powers as an athlete, musican, and elocutionist, but I found that these are not strictly delusions, but exaggerations of undoubted faculties possessed by him. He has, however, true grandiose delusions, for "he possesses a gold mine." Speech somewhat hesitant and tremulous, especially of lip explosives.

After some weeks he improved mentally and physically and nine months after admission he was able to work in the fields five hours a day.

June 19, 1899.—The notes state that he has been having

convulsive seizures, and six weeks ago he had an attack of suicidal mania, attempting to mutilate himself and gouge out his eyes. There is now more dementia indicated by the blank facial expression and his conversation. He does not respond regularly to the calls of nature. Tremor of facial muscles more marked. Beyond walking with a wide base no sign of ataxy. He is dangerous to himself and others.

December 5, 1899.—Patient helpless, bedridden, and demented; death occurred a week later from broncho-pneumonia and cystitis.

Autopsy (summary of notes).—Old scar on glans penis, no bed sores. Skull cap thick and dense. Dura mater adherent to pia-arachnoid over central convolution of left side. Pia-arachnoid of pre-frontal and central convolutions, thickened, opaque and milky. Some wasting of convolutions and cortex somewhat diminished. Fourth ventricle dilated, granular ependyma. Brain—right hemisphere 17½ ozs., left 17½ ozs. Cerebellum and pons—5 ozs.

Cause of death.—Broncho-pneumonia, cystitis and pyonephrosis.

Microscopical examination (summary).—Atrophy or absence of tangential and superradial fibres in various situations of atrophied convolutions, marked vascular congestion, with numbers of plasma cells in perivascular lymphatics. Atrophy and degeneration of small and medium sized pyramids. Glia cell proliferation. *Spinal cord.*—Some leptomeningitis not more marked posteriorly than anteriorly, the same applies to vessels which are congested and their walls thickened. In *cervical region* the degeneration is limited to Goll's column. By Marchi method there are scattered fibres degenerated in all the white matter. In *upper dorsal* and *mid dorsal* regions there is extensive atrophy of root-fibres and their intraspinal projections. In lower dorsal region there is also some diminution of fibres in the comma tract. Lissauer's zone is most affected about the fourth and fifth dorsal, where the roots are most denuded of fibres. About the eighth dorsal, but not above this, there is obvious sclerosis of the crossed pyramidal tracts, which becomes more obvious as we proceed downwards. In the lowest dorsal and lumbo-sacral regions the roots are not so markedly affected and the degeneration in the posterior column takes the characteristic winged appearance, the apex of the wing on either side corresponding to the root zone. There is not a marked atrophy of the fine plexus around the cells of Clarke's column.

A more than usual number of coarse fibres persist in the sclerosed posterior roots. The endogenous fibres in this region are but little affected. At the level of the fifth lumbar segment and extending into the sacral segments is a dilation of the central canal which extends back as far as the posterior surface. This is not an artefact for it is lined with the characteristic epithelium, and represents a congenital failure to close up the canal in this region by the ingrowth of fibres. At the posterior surface and corresponding to the mesial line is an agglomeration of vessels, dense fibrous tissue and *a few bundles of embryonic muscle fibres.* In the fibrous tissue also there are scattered groups of ganglion cells, and small nerve-fibres. The cord in this region, and, indeed, throughout the lower dorsal and lumbar region is very small. One structure, however, is not diminished more than another. It appears to be a failure in development. In a section of the last lumbar spinal ganglion, in what corresponds to the white ramus are a group of cells which stain slate blue with the Weigert method, and from these issue a number of coarse medullated fibres. Whether this is an abnormal condition I am unable to say.

Conclusions are (1) that this patient had thoracic cutaneous anæsthesia, had it been looked for; (2) That the ataxy was not a permanent feature of the clinical symptoms, because there was neither marked atrophy of the fine plexus around Clarke's column, nor was there much atrophy of the lumbo-sacral region, except of the fibres concerned with reflex spinal tonus; moreover the endogenous fibres were not affected in the lumbo-sacral region. Therefore, the ataxy that existed was due more especially to a condition which might under circumstances vary, viz., the reflex spinal tonus and its inhibition by cerebral influence.

The general atrophy of the cord indicates a general nutritional defect; this is frequently met with in these cases of tabo-paralysis, but in this case it might have been congenital.

Case 42.—*Tabetic general paralysis, with characteristic pathological changes affecting the brain, spinal cord, posterior roots, and optic nerves. Marked degeneration of fronto-occipital and fronto-temporal association tracts, causing dilatation of lateral ventricles.*

J. H., aged 37, occupation a tailor, admitted to Claybury, June, 1899.

Abstract of notes.—Married eleven years, three children, two alive, one dead. Wife states that he had fits, and had complained of pains in his legs for two months prior to admission. There was a history of insanity on the father's side.

Mental state on admission.—He is noted as being strange in his manner, wandering constantly from one subject to another, so that it was difficult to obtain from him a coherent account. He states that he hears voices talking to him at night. He has no idea of time or place, and he believes that his wife and family live here. He is under the delusion that he has murdered some of his children by strangulation, and he wants an operation performed on his legs, presumably on account of the pains.

Physical state.—There is marked shortening of the right leg, the muscles of which were very wasted, and there is evidence of old hip disease, (?) Charcot's joint. There are patches of alopœcia on the head, and he states that he has had both gonorrhœa and syphilis. The knee-jerks are absent, and the left leg is retroflexed, jerking and ataxic in movement. The pupils are unequal, the left being dilated and sluggish in reaction to light and accommodation; the right pupil reacts fairly readily. There is well-marked primary optic atrophy on both sides, more obvious in the left eye. Diagnosis at first was mania; towards the end of the year he commenced having seizures affecting especially the right side, the diagnosis of general paralysis was then made. At the end of January, 1901, he died of pneumonia and cardiac failure.

Post-mortem examination.—Body is well nourished, no external marks of syphilis, right leg 3 inches shorter than the left. Skull dense, excess of fluid in subdural and subarachnoid spaces. The pia-arachnoid was thickened and opaque in the frontal and fronto-parietal regions, convolutions were atrophied, though not markedly so; on stripping erosions were seen. The lateral ventricles were granular and dilated. The fourth ventricle was also granular. There was well-marked atrophy of both optic nerves.

Cause of death: (*a*) *Primary.*—Broncho-pneumonia and gangrene of lung. (*b*) *Secondary.*—Tabetic general paralysis.

Examination of spinal cord by Marchi and Marchi-Pal methods showed by the former a number of recent degenerated fibres in the crossed pyramidal and direct tracts, also scattered generally n the posterior columns. The degenerated fibres were seen at all levels of the cord, and were probably therefore mostly belonging to long systems. By the Marchi-Pal method denudation of fibres and subsequent sclerosis with a moderate proliferation of glia

tissue was found in the posterior columns of the spinal cord. In the cervical region this was almost exclusively limited to the leg fibres forming the column of Goll. In the lumbo-sacral region, where the denudation of fibres and subsequent neuroglia overgrowth was most marked, the atrophy of fibres was limited to exogenous systems. The only part of the posterior columns which did not show denudation of fibres was the cornu-commissural zone and the median oval area. The short length fibres of Lissauer's tract, the medium length cerebellar fibres, and the long fibres which together form Goll's column, are in great part destroyed or atrophied. The fibres of the posterior roots in many of the sections show considerable denudation of fibres. In the upper lumbar and lower dorsal region there is still a great diminution of exogenous fibres entering into the formation of the posterior columns, and there is a corresponding overgrowth of glia tissue. The atrophy of the fibre plexus around the cells of Clarke's column is only moderate. *Cauda equina.*—To the naked eye the sections show an obvious difference in the anterior and posterior parts. In the former the roots are of good size, and are stained blue, whereas in the latter the roots are atrophied and unstained. Examined microscopically, the posterior bundles are considerably shrunken, and exhibit very few fibres. Some are apparently almost destitute of fibres, consisting only, or for the most part, of connective tissue. The anterior bundles (motor) are normal in appearance. In both anterior normal and posterior sclerosed roots there are many congested vessels. The walls of these vessels, however, present no marked abnormality.

Microscopic examination of brain.—Marked degeneration of fronto-occipital and fronto-temporal association fibres, atrophy of superficial cell layers of cortex in pre-frontal and fronto-central regions, slight glia cell proliferation, only small amount of vascular change. Atrophy of tangential and supraradial fibres in same situations.

Case 43.—*Tabes of four years standing, then a congestive seizure, followed by transitory aphasia and slowly progressive dementia.*

R. B., aged 50, admitted to Colney Hatch, September 12, 1900. Ticket of leave man, sent from Pentonville, single, occupation tailor, working as a sewing machinist, previously in the

Marines. Admits having had a sore on the penis, which gave him no pain, and he was not treated. Lymphatic glands generally enlarged, papery scars over chest and abdomen.

History of illness.—Four years ago had a cord-like feeling round the waist, he has drunk heavily at times, but there is no marked history of alcoholism.

Family history not obtainable from friends, as he has no visitors. He himself is unable to give any satisfactory data. He was transferred from Pentonville with a certificate, indicating that he had had a congestive seizure, followed by transitory aphasia, with dementia, and wet and dirty habits.

Mental condition.—He has no actual delusions, he is a little incoherent in his answers, but can give a fairly intelligible account of his past life, although his memory as to place and time is defective, for he does not know the year, nor the month, but he was able to tell me that he was in the Marines, and that he had been in Pentonville prison, where he was sent for having stolen a piece of meat. He can hardly do simple multiplication sums, and if able to give a correct answer, he is quite lost when the figures are reversed.

Physical condition.—There is a loss of expression of the face, especially of the right side, the right grasp is a little feebler than the left. The speech is very slightly hesitant, no tremor of tongue or lips. He has a very ataxic walk, and he cannot stand with his eyes shut. He has pain in his legs, which he thought were rheumatic; he has suffered with them for twelve months, and thought that they were due to the machine work. Right pupil irregular, rather smaller than the left (3mm.), inactive to light, they react to accommodation. He has some loss of sense of position in the hands and feet, he can correctly locate light tactile sensations, and he recognises the prick of a pin over the whole body. The leg can be raised to a right angle with the body, indicating a fair amount of hypotonus. The knee-jerks are absent on both sides. Examination of the fundus reveals nothing abnormal on either side.

June, 1901.—There is very little change in the condition of this patient. He is somewhat thinner, but his mental condition has not altered for better or worse. He walks more unsteadily and there is a tendency to fall to the right. The tongue is more tremulous and protrudes towards the right slightly. The pupils are unequal, the right 3½ mm. irregular, the left 3 mm., also irregular, inactive to light, react sluggishly to accommodation.

Case 44.—*Tabes eleven years after infection. Ataxy, sensory disturbance, bladder disturbances, history of monetary troubles, development of mental symptoms, disappearance of spinal symptoms in great measure. Knee-jerks absent on admission to asylum. Mental and physical improvement. Relapse, development of left-sided seizures terminating in hemiparesis without any contraction. Reappearance of knee-jerk on left side. Attack of dysentery, recovery. Attacks of vomiting (probably crises). Further left-sided seizures spreading to right side. Death two years after onset of mental symptoms. Right hemisphere* 17¼ *ozs. Left* 19¾. *ozs. Naked eye gray degeneration of posterior columns of spinal cord, also of pyramidal track on left side in dorsal and lumbar regions.*

W. H., college porter, Guy's Hospital, aged 38. Married, two children, and two miscarriages; contracted syphilis in 1888, for which he was well treated. No history of insanity in the family, not intemperate. In May, 1899, complained of pains in the legs and unsteadiness of gait. He was admitted into Guy's Hospital under Dr. Pitt, to whom I am indebted for the following notes. Pains in the lower extremities, in the bones and muscles rather than in the joints. He complained of unsteadiness in walking, and he noticed diminution of sensibility across the buttocks and scrotum, and along the inner sides of the thighs. Thinks when he is sitting down that he is on a pole 3ins. in diameter. Marked ataxic gait, even with eyes open. Cannot stand with feet together and eyes shut. Knee-jerks absent. Plantar reflexes absent, abdominal present. Impaired sensation of soles of feet and inner sides of thighs. Pupils equal, small A.R. Speech normal. At times depressed, memory and intelligence good. Frequently he has difficulty in starting micturition, and is troubled with constipation. Frenkel's treatment applied with benefit; discharged in unchanged condition.

His wife relates that he had saved £350 in eleven years, and he lent some of it to a friend who did not repay him, and this caused him great worry and anxiety, followed by sleeplessness and depression; his mind became affected, and he was admitted to Bethlehem in September, 1902, and to Cane Hill Asylum, December 31, 1900. His condition in February, 1901, was as follows:—

Physical condition.—Facial expression denotes mental enfeeblement; there is some obliteration of the lines of expression, especially on the left side. The left eye is rather more open than the right. There is marked tremor of the tongue and lips, and

staccato speech ; there is more tremor on the left than the right side. Right pupil, 2 mm., left, $2\frac{2}{3}$ mm. ; right, accommodation to $1\frac{2}{3}$, and left to 2. No reaction to light or pain. Gait slightly unsteady and incoordinate, rather a wide base, no stamping of feet, no Romberg symptom, no definite area of affection of cutaneous sensibility to pain or touch anywhere (answers quite reliable) ; sometimes confuses a touch with a prick. No loss of sense of position of joints. A needle introduced deeply into the muscles of the legs in different regions, and the patient made to move the muscles, exhibited a loss of muscular sense. Plantar reflexes normal, all other superficial reflexes brisk. Knee-jerks, and triceps-jerks absent, hypotonus of hamstring muscles extreme (30 degrees beyond the vertical). No crises, no loss of control of sphincters, and no difficulty now with the bladder or bowels.

Mental state.—Very emotional, readily laughs or cries. Marked *bien être*, weak-minded and childish in conversation. Comprehension fairly good, likewise orientation. He can give a fairly good account of his past life, and he has no delusions, illusions, or hallucinations.

For some time he improved both mentally and physically, then in April, 1902, he suffered with left-sided seizures and vomiting, which preceded an attack of acute dysentery ; blood and slime in stools, pyrexia and distended tender abdomen. It was a month before the acute symptoms subsided, and two months before he was convalescent. During this period of convalescence he suffered with paroxysmal attacks of vomiting (crises ?) on several occasions. In August he was much more demented, and was the subject of many epileptiform seizures, mainly affecting the left side, leaving him in a nearly hemiplegic condition ; whether he was hemianæsthetic could not be determined, as he did not speak when questioned.

The *left knee-jerk was now obtainable*, the right still absent.

August 29.—He commenced a succession of epileptiform seizures affecting especially the left side (head and eyes turned to the left). Temperature subnormal. August 31.—The convulsions on left side ceased, the limbs were flaccid and helpless. Convulsive spasms of the right side now occurred, conjugate deviation of head and eyes to the right. Temperature 100·6°, quite unconscious ; death supervened on September 1.

Autopsy (abstract).—No scar on penis or glands or signs of syphilis on the body. All the viscera were apparently healthy, with the following exceptions. *Heart.*—12 ozs., cavities dilated, stopped in diastole, muscular substance greasy and friable. No

valvular disease. *Lungs.*—Healthy, excepting hypostatic pneumonia of both bases. All the abdominal organs, like the thoracic, showed signs of congestion, the result of asphyxia from prolonged seizures. Throughout the large intestine there was evidence of the attack of dysentery from which he had suffered. The mucous membrane was thin, and showed white lines and small patches indicating scar tissue, the solitary follicles were in many places prominent, but everywhere the condition denoted that a healing process had occurred, and there were no signs of recent affection of the internal coats of the bowel. *Brain.*—Excess of fluid, marked thickening of pia-arachnoid, especially over fronto-central convolutions, great wasting of right hemisphere. Weight, right, 17¼ ozs. Left 19¾ ozs. Great dilatation of right ventricle, especially in posterior part, due to atrophy of brain substance, forming floor and outer wall ependyma granular. Convolution, pattern complex and good. Brain kept for investigation, fourth ventricle very granular. Spinal cord, obvious atrophy of posterior roots, and gray degeneration of posterior columns extending from the seventh and eighth cervical segment downwards. Degeneration of left crossed pyramidal tract in lower dorsal and lumbar regions. Examination of blood contained in the femoral vein showed a great abundance of choline.

Microscopical examination.—Brain exhibited marked cell and fibre atrophy, glia proliferation, and vascular changes, with plasma cells.

Case 45.—*Tabes of four years duration in a man aged* 55. *Arteriosclerosis affecting the small cerebral arteries especially, marked atrophy of the right hemisphere, with multiple miliary softenings, left hemianæsthesia, and hemianopsy.*

C. C. Occupation, labourer and turner at the Arsenal; afterwards railway porter, and for thirty years a signalman. Aged 55. Married twenty-seven years, no children; wife had two premature births. Drank somewhat when a porter, but not recently. His symptoms began three or four years ago; he noticed difficulty in passing his water, which ran slowly, and he continually wanted to go, but the urine only dribbled away; also, he had difficulty with his bowels, and shooting rheumatic pains in the left leg. He gives a history of having had a sore on his penis four years before marriage. He was treated for three or four months at the Royal Free Hospital. He had secondary symptoms, sore throat, rash and hair falling out. He was admitted to Cane Hill Asylum with delusions of persecution. No insanity in the family.

On October 4, 1900, he had an epileptiform seizure, which left him in an incoherent mental condition, and he could give no rational account of himself.

Physical condition (October, 9, 1900).—He is in fair state of nutrition, and was able to give a good account of himself and his illness, which I subsequently found reliable. He is a well-built man with several papery scars over the back. He has an ataxic gait, the left foot and leg being most affected. The knee-jerks are absent on both sides even with reinforcement. There is marked hypotonus in both legs. Romberg's symptom is present; he has not suffered with any crises, and he complains of no visceral symptoms, except the difficulty of making water. The pupils are equal, perhaps the right a little larger than the left (2½mms.), inactive to light, react to accommodation. He has complained of imperfect vision in the left eye, and tested roughly, he is found to have left-sided hemianopy. He has had one left-sided epileptiform seizure since he has been in. He says sometimes he will wake up and not know that he has a left leg and hand. He has been unable on account of the numbness to button his clothes with his left hand for four or five months. There is great incoordination in movements performed with the left hand. He cannot touch the tip of the nose, but he can do it with the right hand. He says he cannot feel his moustache with his left hand, but he tells me of his own accord that he has no loss of power, and he certainly has a good grasp. His sensation was tested, and the accompanying charts indicate the condition found. It may be remarked, however, that although it took one hour and a half to make these charts, they can only be considered approximately true, for, as in all cases, response to stimulus varies; but subsequent notes made by Dr. Gilfillan show a marked difference in sensibility on the two sides of the body. Very possibly, the hemianæsthetic condition may vary in degree according to variable conditions of the opposite hemisphere, but the *post-mortem* results absolutely accord with this condition. I saw the patient several times subsequently. I did not notice any speech affection or tremor of the lips and tongue, and I looked upon the disease as a case of tabes with mental symptoms. There was only a very mild degree of dementia, his knowledge of time and place and memory for ordinary events of his past life seemed fairly good. The attendant states that he is dull, confused and incoherent after he has had fits.

December 15, 1900.—Had a slight seizure on November 30, followed by increased weakness of left leg. Had a seizure on

December 13, and another to-day, affecting his left side. Is now confined to bed.

February, 1901.—Knee-jerks absent. Plantar reflexes exaggerated. Left leg paralysed, left arm weak (patient says it is senseless). Sensation right side good, whenever roughly touched on left leg, refers touch to inner side of thigh; touched on left arm, refers touch to point of shoulder. Complains of pain and numb-

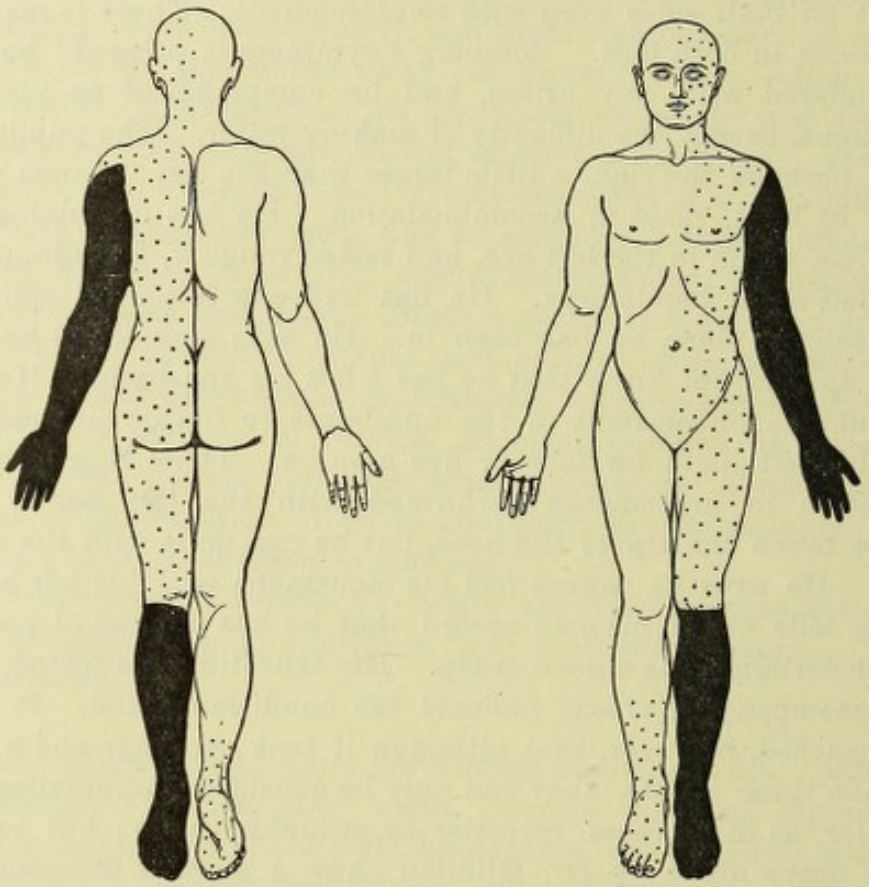


FIG. 31.

ness of left leg. Pain in left hip shooting down to knee and calf. Pain during passive movement of left leg.

Girdle pains when up, never in bed. Shooting pains in left arm. Pupils equal. Left reacts to light sluggishly, both to accommodation. Tongue steady. Right side distinguishes hot and cold. Left side cold resembles pricking, and hot not recognised. (Dr. Gilfillan.)

March 21, 1901.—Had a seizure to-day affecting the left side.

April 17.—Has marked dulness over left lung, and impaired breath sounds. Temperature hectic in type. Feeble health.

May 1, 1901.—Small râles can be heard over both lower lobes, but the sounds are muffled in the left. Is rapidly becoming much worse.

May 7.—Gradually sank and died.

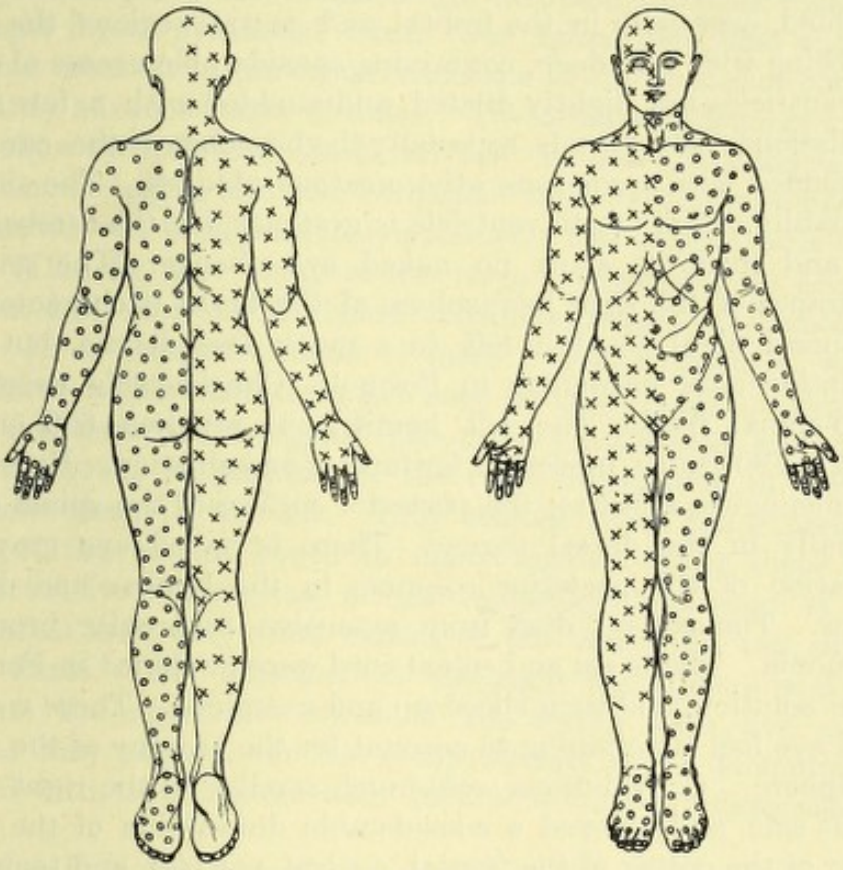


FIG. 32.

= Thermo-anæsthesia

= Hyperæsthesia

Cause of death as certified.—General paralysis of the insane. Tubercular broncho-pneumonia.

Abstract of post-mortem notes: Body well developed and well nourished, except thighs and legs, which are wasted and flabby; a large amount of subcutaneous fat present in all parts of the body. No marks of syphilis. *Skull.*—Nothing abnormal. *Membranes.*—Dura mater thickened and firmly adherent to skull

along the vertex. Pia-arachnoid extremely opaque, much thickened, chiefly in frontal, central, and parietal regions. In places it is somewhat maroon coloured, probably the result of old, slight hæmorrhages. The membrane strips readily without causing decortication. Pacchionian bodies are very large. *Spaces.*—Subdural contains excess of clear fluid; sub-arachnoid, excess of fluid, which is slightly blood tinged. *Brain.*—The left hemisphere weighs 4 ozs. more than the right; the convolutions of the right hemisphere are of normal complexity, but markedly atrophied, especially in the frontal and central regions, the sulci here being wide and deep, containing considerable excess of fluid. The ventricles are slightly dilated, and studded with a few small granulations. The vessels, especially the branches of the carotids, are studded with numerous atheromatous plaques. The sinuses are healthy. The fourth ventricle is granular, but the cerebellum, pons and medulla, show no naked eye change. The general appearance of the right hemisphere of the brain is characteristic of general paralysis, the left to a much less degree, but *vide* description after hardening in Formol. Considerable excess of cerbro-spinal fluid. The left hemisphere weighed 600 grams, the right 490 grams, preserved for further examination. Extensive leptomeningitis affecting the posterior surface of the spinal cord, especially in the dorsal region. There is naked eye gray degeneration of the posterior columns in the lumbar and dorsal regions. The patient died from extensive tubercular broncho-pneumonia. The brain and spinal cord were hardened in Formol-Müller solution, and then sliced up and examined. There was no naked eye foci of softening to account for the atrophy of the right hemisphere. Convolutions were much smaller on the right than the left side, and showed a considerable diminution of the gray matter of the cortex of the frontal, central, parietal, and temporal regions, where there was very marked thickening of the pia-arachnoid. The right lateral ventricle was markedly dilated, especially in the posterior part, and extended farther back than the left.

Microscopical examination.—By Nissl, Marchi, and Weigert methods demonstrated the following facts. There was a random degenerative process due to vascular changes, which was the principal cause in the difference of weight in the hemispheres. The fact that no gross change could be seen, and yet there was so much atrophy, accords with the microscopic observation of numerous scattered miliary areas of softening in the white matter immediately subjacent to the gray cortex, affecting especially the

radial fibres of the ascending parietal, and the optic radiations about the calcarine fissure. Sections through the ascending parietal and ascending frontal show a gross old degenerative change of the ascending parietal. This accords with the fact that there was a hemianæsthesia without much loss of power when I saw him. Later, no doubt, there was extensive destruction of the ascending frontal, or more likely some part of the path of the fibres on their way to form the pyramids of the medulla, for the left pyramid presented a naked eye atrophic sclerosis, likewise the right direct pyramidal tract. The spinal cord also showed atrophic sclerosis in the usual situation in the posterior column of a fairly advanced case of tabes. The miliary softenings in the brain show the following characteristics: dilated congested vessels with hæmorrhages into the lymphatic sheaths, accompanied by endothelial cell proliferation lymphocytes and plasma cells (*vide* Plate VIII., fig. 2). Many of the larger neoplastic cells contain pigment derived from the blood in various stages of disintegration. In the neighbourhood of these vascular changes, which often have a dendritic form are seen ganglion cells undergoing acute degenerative changes. The miliary softenings are much more numerous in portions of the right hemisphere in various situations than in the left. They seldom affect the superficial layers of the cortex, hence in many regions where there is a very extensive destruction of the radiating fibres there is less destruction than one would expect of the tangential and superradial fibres. The affection of the right hemisphere and the fact that the superficial fibres of the cortex were not much affected may account for the comparatively slight dementia he suffered with, as compared with the amount of brain destruction.

Case 46.—*Publican, formerly policeman, suffered five years with progressive tabes, development of mental symptoms, becoming lustful, suspicious, extravagant, and dangerous to himself and others. Speech affection developed, progressive dementia with intervals of improvement, epileptiform seizures, death. Advanced tabic lesion of the cord, chronic and acute degenerative changes of the brain. Heterotopia spinalis.*

W. A. McSt., publican, aged 42. Married; no children. At 17 joined the Guards, served eight years, became colour-sergeant. While in the army had gonorrhœa and sore. At 25 joined the police force, served thirteen years, invalided with locomotor ataxy. First symptom noticed was sudden loss of power in his

legs, while serving notices as a jailor. After this suffered with pains in his legs and girdle sensations, no bladder trouble until recently; he has not suffered with headache nor any visceral crises, does not complain of his food.

Physical condition.—Ataxic gait, Romberg's symptom marked, triceps and knee-jerks lost, some loss of sense of position in hands. Right pupil 4 mm., left 5 mm., irregular, and neither react to light, but sluggishly to accommodation. He stands with a wide base and with the support of a stick. He has some difficulty in maintaining his position on the polished floor, and he finds a special difficulty in rising from a sitting posture. He has a typical ataxic walk, the body being bent forward, the neck stiff, but slightly inclined down, so that he can watch and control the movements of his legs. The advancing leg is extended at the knee instead of being flexed, jerked forwards and outwards at the hip, the heel being brought down with a stamping action.

Expression.—He has a depressed and anxious look when not engaged in conversation, but he is easily moved to tears or laughter. When speaking there is a marked tremor in the lips noticed, and there is a tremor in the tongue on protrusion. His speech, like his handwriting, is hesitant, tremulous, and many of the syllables are slurred.

Skin sensibility.—Over the thorax, from the sixth to the tenth segments inclusive, there is a loss of sensibility to light tactile impressions. Above and below this region, he feels light tactile sensations, but there is often some confusion as to localisation, and on the peroneal surface of the legs and the soles and dorsal surfaces of the feet, light tactile sensibility is considerably diminished, and often the stimulus is either not felt or incorrectly localised in these regions. Painful sensation is blunted considerably over similar regions to those in which tactile sensibility was impaired or lost. Very often there was a considerable delay (two or three seconds) before he responded to the stimulus. In the regions above and below the areas of analgesia pricking seemed to cause unusual burning pain, so that there was hyperæsthesia over the inner side of the leg and thighs and lower part of the abdomen. I could detect no thermal-anæsthesia over any part of the body (*vide* charts of sensory disturbances).

Reflexes.—Triceps and knee-jerks absent; superficial reflexes—plantar present, but not active, cremasteric present, fairly active. He has had no difficulty with his bowels or bladder, he has no loss of control. There is some degree of hypotonus, but not in proportion to the ataxy. The muscles are firm and strong, leg

extended can be brought to 90° with body. From his conversation, one would judge that he had suffered from satyriasis.

Vision.—No failure to recognise colours, he has recently complained of dimness of vision of the right eye. There is commencing optic atrophy, affecting especially the temporal half of the disc, and apparently some cupping, vessels normal, the fundus has a gray appearance in this situation, and the vessels are curved. There is no myopic crescent.

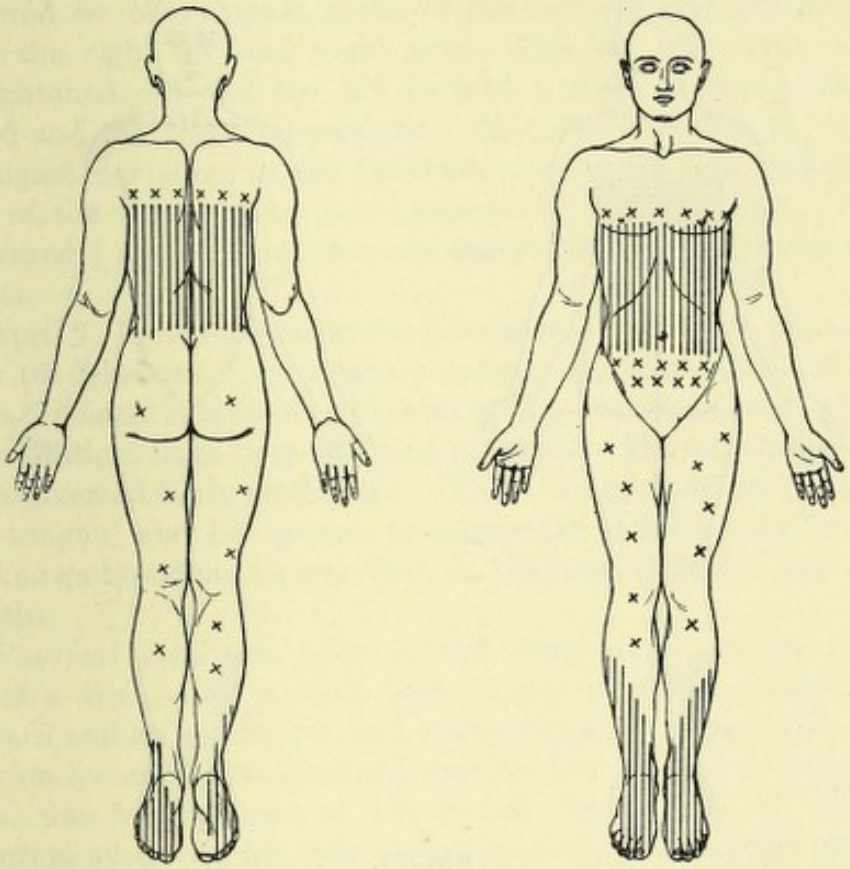


FIG. 33.

= Hypalgesia = Hyperæsthesia

Mental condition.—He is able to converse on most subjects, and to give an account of his life, which accords fairly well with what his wife had told me. He has a fair knowledge of time and place, but he has delusions about his wife's fidelity or her father's accusations against him on this score. He has moderate grandiose delusions about his property, and what he can do when he gets out. From his conversation he has evidently been leading

a very loose and extravagant life, throwing his money away at music-halls and otherwise ; still, one recognises that in all he says there is a certain amount of truth and reason mixed with folly and irrationality.

History (from wife and sister).—Wife had no children ; she herself has good health, he was a good, kind, and affectionate husband. He suffered for five years with locomotor ataxy. Up till six months ago he was mentally quite right, then she noticed

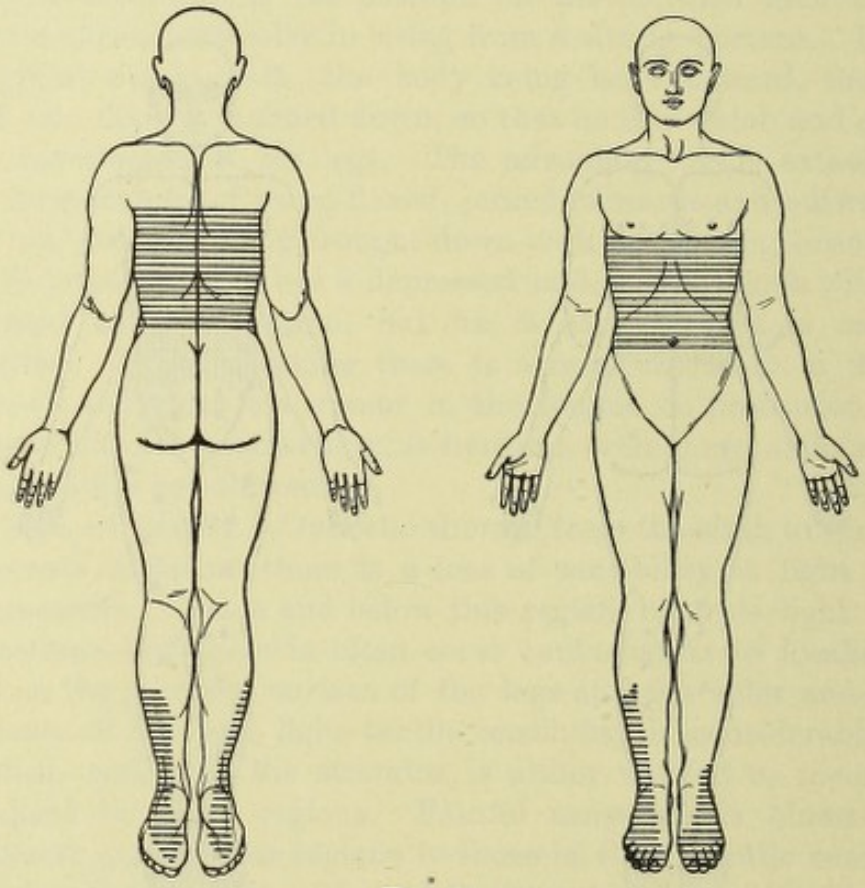


FIG. 34.

= Cutaneous Anæsthesia

he became lustful, irritable, sleepless, given to extravagance, and planning all manner of schemes; lately has become suspicious, jealous, and at times violent, threatening to murder people, and having delusions about his wife's fidelity. This is indicated in the letter which he has written, which shows both dementia and delusions, also characteristic tremor, and leaving out of words

and syllables. *His wife noticed that since his mind became affected he had become much stronger in his legs.* She made this remark without any leading question being put.

March 2, 1901.—He suddenly fell down, Dr. Bolton who saw him describes the fit that ensued as follows. Marked clonic spasms of the right side of the face, less marked to the left. Right arm lying still. Left arm occasionally moved, apparently voluntarily as the patient pointed towards his head. Then the right arm was convulsed and next the legs, the right being more affected than the left, a tonic left-sided spasm then occurred followed by clonic movements of the left leg and left arm, and later the right leg and right arm. The left arm next became straightened out and the left bent at a right angle, the left leg flexed and the right leg straight. This was followed by marked conjugate deviation of the head and eyes to the left, and the left side of the face became more convulsed than the right. Pupils measured 7 mm. There was no ataxy noticeable in these movements.

April 7, 1902.—Came to the laboratory. Mentally he is much freer of delusions. He appears in better physical health and the acute maniacal symptoms which he had on admission, accompanied by exaltation, have in great part subsided. He remembered me, and conversed fairly intelligibly. There is slight tremor in the lips and tongue, and his speech is somewhat syllabic and hesitant. He knows how long he has been at Claybury and the day of the month.

Physical condition.—He is still very ataxic, walks by the aid of a stick, with a wide base; knees extended, head looking forward and on to his feet, and stamping gait. Lying flat on his back on a couch it was noticed that he *had partial footdrop both sides*, due to weakness of the dorsal flexors; by an effort of attention even with his eyes closed he could dorsally flex the foot, but if his attention were taken away, the foot would fall back again. When told to flex the knee and hip simultaneously, the failure of the dorsal flexion was noticeable, when told of it he immediately tried to correct it, and this he could do by an effort of attention, but it was noticed that the correction did not come till after the flexion of the hip and knee had taken place, and an appreciable interval elapsed; owing to the fact that it was brought about entirely by cerebral impulses.

Sensation (subjective).—He complains of numbness on the soles of the feet, and when tested there is manifested some hypæsthesia and hypalgesia of the feet on both sides. There is a

complete loss of sense of position in the toes and ankle joints, and also to some extent of the knee-joints. There is considerable hypotonus of the hamstrings on both sides.

Thoracic sensibility.—This seems to have changed somewhat since I last examined him. I can now find no definite anæsthesia of the left half of the thorax in front, corresponding with the anterior divisions of the roots, but over the posterior divisions I find loss of sensibility to light tactile impressions over the fourth to seventh segments. The right half of the thorax the same condition obtained posteriorly, but in front a very curious condition existed; either he did not feel at all light tactile impressions over the front of the chest, extending from the fourth to seventh segments, or if he were touched more heavily he invariably pointed to a corresponding point on the left side of the thorax, (allochiria). These observations were quite reliable. He does not now show any hyperæsthesia. The epigastric reflexes were present on both sides also the cremasteric. The plantar are absent, there is no marked incoordination in the hands, but there is some loss of sense of position in the joints. There is no impairment of tactile sensibility. The pupils measure, left 6 mm., right 5 mm. A week ago he had paresis of both external recti; this, however, is now passing off. Three weeks ago he had an attack of profuse diarrhœa which Dr. Bolton considered was of the nature of an intestinal crisis.

Patient improved and tried to make himself useful until July 15 when he had a succession of seizures which terminated fatally in twenty-four hours from commencement.

Post-mortem notes (abstracted).—Well nourished. Scar in region of frenum. Dura mater, intensely congested, otherwise natural. Subdural spaces.—Great excess of fluid, well over tentorium. Pia-arachnoid.—Considerable fronto-parietal milkiness and thickening, marked pial extravasations in the postero inferior part of the external surface of the right hemisphere, slight ditto left hemisphere. The whole encephalon is intensely congested. Weight 1,415 grammes. Right hemisphere 632 grammes, left 590 grammes. Left hemisphere more simply convoluted than right. Fourth ventricle.—Covered with very fine granulations, most marked in calamus. Heart.—Dilated and flabby. Valves.—Mitral shows slight atheroma. Coronary arteries.—A little early atheroma. Aorta and great vessels.—Marked atheroma in all stages, mixed calcareous and pearly fibrosis. Abdominal aorta.—Markedly atheromatus.

Cause of death.—Cardiac failure and general paralysis.

Microscopical examination (by Nissl method).—*Brain, left hemisphere* : *First frontal.*—Marked vascular congestion, perivascular lymphatics filled with proliferated plasma cells. Disordered Meyert's columns, acute cell changes; glia cell proliferation; all cortical layers affected. Vascular changes more marked in the deeper layers. Moderate increase of spider cells in superficial layers of cortex. Many of the small, and a fewer number of the medium sized pyramids, distorted in shape. The cell changes appear to be more marked in the polymorphic and granular cells of the deeper layers. Coloration of protoplasm of many of the cells suggests coagulation necrosis.

Second frontal.—Changes similar but very much less intense. There is obviously an association between the vascular and acute cell changes in the first frontal.

Lower part of ascending frontal and parietals and Broca.—The same changes as above, only more marked. The small and medium-seized pyramids are more affected. The perivascular lymphatic cell proliferation is very marked, especially in the deeper layers. The cells do not present the appearance of experimental anæmia produced by ligation of four arteries with recovery, but much more the appearance of a coagulation necrosis, such as is produced by toxic agents, a fatal dose of abrin or ligation of arteries without re-establishment of circulation. The protoplasm of the cells stains a uniform dull diffuse purplish blue, instead of a bright blue. The medium and small-sized pyramids in this respect, contrast with the large Betz cells, the majority of which show normal Nissl-granules. Pappenheim's stain shows abundance of plasma cells around vessels, and some are found in perineuronal spaces (*vide* figs. 3, 5, 6, Plate VIII.).

Top of ascending frontal.—Very little vascularity and congestion or perivascular cell proliferation. Small and medium sized pyramids deficient, much more so in some places than others. Betz cells numerous, the majority appear fairly normal. In some the nucleus is large, clear, and swollen, others show deficiency of chromophilous substance. The small and medium pyramids are distorted, and processes broken off.

Spinal cord.—Naked eye degeneration in usual situation and amount of the sclerosis corresponds with the second stage of ataxy in the cervico-dorsal and lumbo-sacral regions. Gray matter, lumbo-sacral region; marked vascular stasis probably associated with epileptiform seizures. No great excess of leucocytes around vessels or in contained blood. Neuroglia cells, small variety in abundance. Ganglion cells of anterior and posterior horn, apparently normal in numbers. Anterior horn cells are

rather swollen (probably acute change from venous congestion), nucleus large and clear. Nissl-granules in cytoplasm, and in processes fairly abundant and normal in tint and shape. Cells of base of posterior horn and substantia gelat. apparently in numbers normal. I am unable to judge whether the protoplasm of the cells is normal. The slight cell changes that exist are probably due to the venous congestion occasioned by the seizures in which he died. The glia cells of the degenerated posterior columns are mostly small round or oval, there are some few Deiter's cells more than elsewhere, also granulation corpuscles and excess of glia fibrils.

Microscopical examination of the central nervous system for acute degeneration (by Marchi method).—*Cerebrum.*—Various portions of the brain which were examined by Nissl method were also examined by the Marchi method; no recent degeneration was found in the superficial layers of the cortex. A considerable number of degenerated radial-fibres were found in the ascending parietal, but very few in the ascending frontal. This accords with the fact that very few degenerate fibres were found in the pyramids of the medulla oblongata and in the antero-lateral columns of the cord. In the medulla a well-marked degeneration was found in both ascending branches of the fifth nerve, there was considerable vascular congestion and numbers of black points due to degenerated collaterals in the cranial nuclei of the fourth ventricle, and the cells of many of the nuclei were stained black. That the degeneration of the ascending branches of the fifth nerve was genuine was shown by the fact that the adjacent restiform bodies were quite free of black particles and granulation corpuscles.

The spinal cord exhibited very little recent degeneration, but the fifth cervical segment showed a number of recently degenerated fibres on one side in the postero-external column, especially in the cornu-radicular and root zones. This to a less extent was seen in the remainder of the cervical segments below the fifth. The examination of the dorsal segments showed a marked dilatation of the central canal, normal epithelium lining the whole canal, and, therefore, it was a true heterotopia (*vide* Plate IV.). Around the dilated canal were proliferating, round glia cells. Both the epithelium cells lining the central canal and the glia cells are filled with black granules, products of degeneration, thus showing that they have an active phagocytic function. A further description of the epithelial and glia proliferation is given on p. 293.

Microscopical examination for chronic degeneration (by Weigert

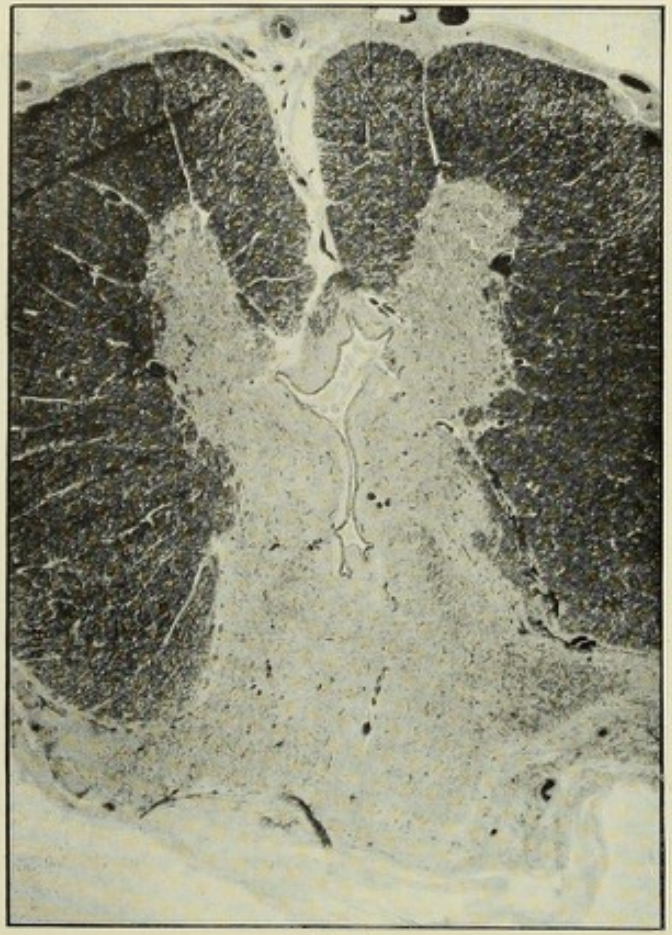


Fig. 1.

Heterotopia spinalis seventh dorsal segment. The irregular dilated central canal lined by ciliated epithelium is shown. There is obviously a very marked glia cell proliferation, for although the fibres of the posterior column are for the most part destroyed, yet the posterior column bulges backwards beyond the normal; usually when there is such advanced tabes as this the posterior column is flattened and retracted. Magnification

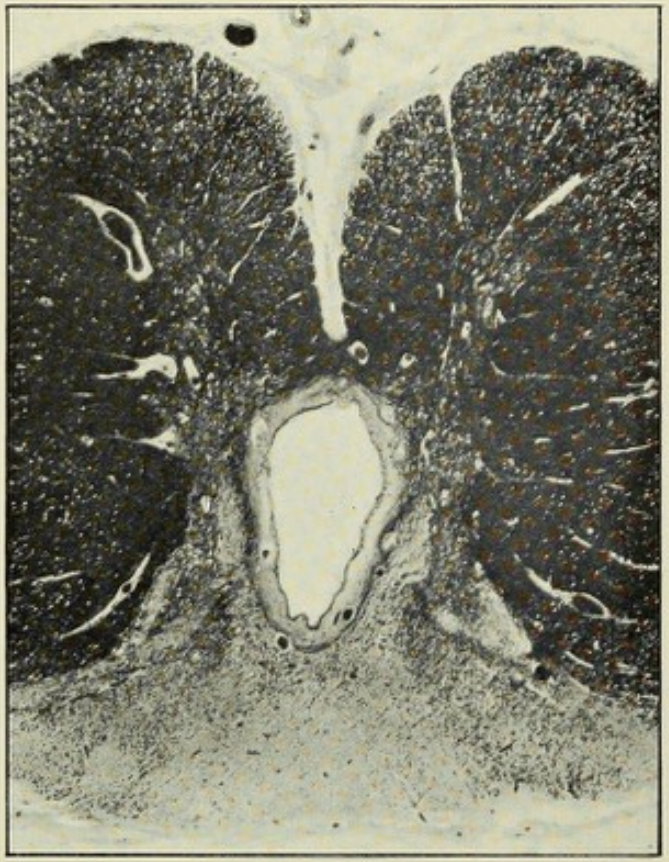


Fig. 2.

Dilated central canal lined with columnar epithelium. The column of Clarke lies on each side, it is oval shaped; the fibre plexus is absent, but bounding it internally and externally are two tracts of fibres, which proceed back from the base of the anterior horn to the cornu-commissural and postero-external tracts respectively, and are in all probability endogenous in origin. Magnification 8 diameters.

and Marchi-Pal methods).—The same regions of the brain were examined, and a marked atrophy of the tangential and supra-radial fibres was found in the ascending frontal and second frontal, very much less in the ascending parietal. There was complete loss of these fibres in the orbital and prefrontal convolutions, and in these regions there were a large number of branching glia cells.

Spinal cord.—There was very marked degeneration of the root fibres and their intramedullary projections extending from the fourth cervical downwards to the termination of the cord, but the degeneration was most advanced in the lumbo-sacral and mid-thoracic regions. The endogenous fibres were in this case markedly affected, there was a great diminution of the fibres of the comma tract, the posterior internal zone, septo-marginal tract, and the oval area of Flechsig, likewise the small median triangle of the sacral region. A considerable number of fibres have disappeared from the cornu commissural zone and been replaced by glia tissue, but this portion of the posterior column is the least affected. Owing to the heterotopia in the dorsal region, the course of the endogenous fibres can be more distinctly seen than in other spinal cords of an equally advanced degree of tabes, with posterior column atrophy and sclerosis. Fibres can be distinctly seen passing from the base of the posterior horn round Clarke's column to form the posterior commissure, decussating with similar fibres from the opposite side. These fibres are of endogenous origin, as they are of fine texture, and can be seen starting from the region mentioned, while the fibres of the root zone are entirely absent, and therefore cannot give origin to them (*vide* fig. 2, Plate IV.).

Teased preparations of the posterior roots attached to the third, fourth, fifth, sixth, seventh, and eighth cervical segments were made. The root-fibres of the third segment were mostly small or medium-sized medullated fibres; they showed no neurilemmal nuclear proliferation. The roots of the fifth showed a number of large medullated fibres with well-developed myelin sheath, and no nuclear proliferation, but there were also a large number which showed the myelin sheath in various degrees, attenuated, and these exhibited a marked nuclear proliferation of the neurilemma. Again, in some there was complete absence of the myelin, and apparently only the neurilemma, the cells of which had undergone marked proliferation, was left. Below this level the rootlets contained only a few solitary myelinated fibres, and in these the sheath was much attenuated.

Case 47.—*Tabo-paralysis, grandiose delusions, ataxy. Duration of disease said to be one year.*

W. J. W. Occupation, labourer. Aged 31. Admitted to Claybury Asylum, November 18, 1895, under the following certificate: "He is strange in his manner, and varied in his moods. At one time he is depressed and pre-occupied, and at another laughs, sings, and whistles. Declares he is a great fighting man, and states that he is the possessor of a gold belt worth £11,000, awarded to him for his successes as a pugilist. He also asserts that he has been a soldier in India, and has fought many battles, and has obtained three medals for the same (although I am informed that he has never been abroad). He further tells me that he is going to buy some land and build houses upon it, which he proposes to let free of charge to the poor. He is wet and dirty in his habits, and his memory is is impaired."

Condition on admission.—Pupils equal. A. R. irregular, gait ataxic, knee-jerks abolished. Scar on penis. Symptoms commenced seven months before admission, probably the cord symptoms of longer duration.

Mental condition.—As above. He is irrational and incoherent.

December 7, 1895.—He is noisy night and day, very mischievous, pulls himself up by the blind cord. Grandiose delusions persist.

January 15, 1896.—Advanced general paralytic. He has had one or two slight seizures. He is very paretic, and in poor general health and condition.

February 8, 1896.—Died of exhaustion of general paralysis.

Autopsy.—Convolutions of the brain are well formed, and are not wasted; the pia-arachnoid is slightly thickened, but it is not adherent to brain substance. Lateral ventricles are normal, the fourth is dilated and slightly granular in its lower part. The posterior columns of the spinal cord are wasted; the aorta is slightly atheromatous at its base. There is hypostatic congestion of the bases of both lungs. On the penis there is an old scar.

Microscopical examination.—First frontal convolution showed some early changes characteristic of dementia-paralytica. The tangential and supraradial fibres were greatly diminished, some increased vascularity, atrophy and changes of the superficial pyramidal cells, and distortion of Meynert's column.

Spinal cord.—The cervical, thoracic, and lumbo-sacral regions showed advanced sclerosis and atrophy of fibres in the usual situations. There is some atrophy of the endogenous systems,

the least to be affected being the cornu commissural zone. The posterior roots are markedly atrophied, only a few myelinated fibres being left in the lumbo-sacral regions (*vide* fig. 39). In the lateral and anterior columns there are diffuse scattered degenerated fibres, but there is no sclerosis. There is marked vascular congestion throughout the cord and medulla. In the sclerosed areas there is an obvious thickening of the vessel walls. The appearances of the cord lesions would indicate tabes of some years standing. The notes which are taken from the case book are too brief to be of much value.

Case 48.—*Tabes of four years' duration, onset of mental symptoms of general paralysis, admission to asylum, epileptiform seizures, attacks of vomiting, death about one year later.*

W. T., aged 38, single, tinsmith, under the care of Dr. Head, to whom I am obliged for allowing me to make an abstract of his notes. Admitted to London Hospital June 5, 1897.

Personal history.—Chancre at 22, which ate away the frenum, followed by repeated ulcerated throats, noticed no rash. He has had pains in his legs and stomach for eighteen months. Last year he had double vision, which lasted six months before it began to improve. Very tired when walking, no difficulty with his bladder, except that he cannot hold his water so long as he used to be able to. About eighteen months ago he became lustful, very different from what he had been before. He had plenty of power, but for the last six months he has become very feeble, but desire is still present, but erection fails. No trouble with the rectum, or other illnesses. He has always earned good wages, has drunk a good quantity, but never enough to incapacitate him. There is nothing noteworthy in his family history, with the exception that one brother is asthmatic.

Nervous system.—Speech good, no attacks or fits, no headache, stands perfectly steady, with the feet together and the eyes open. With his boots on and his eyes shut, no swaying movement is noticed, but these come on when he takes his boots off. He can rise from toes to heels quite easily with his eyes open, but he tends to fall forwards when his eyes are closed. Very slight incoordination of left hand, with eyes shut on touching the nose, no paresis. He complains of rheumatic pains in both knees, a tired, aching feeling, but not shooting pains. Cutaneous sensations in all forms both in time and place are excellent. Knee-jerks are present on both sides without reinforcement, the right greater than left. On a previous occasion the left

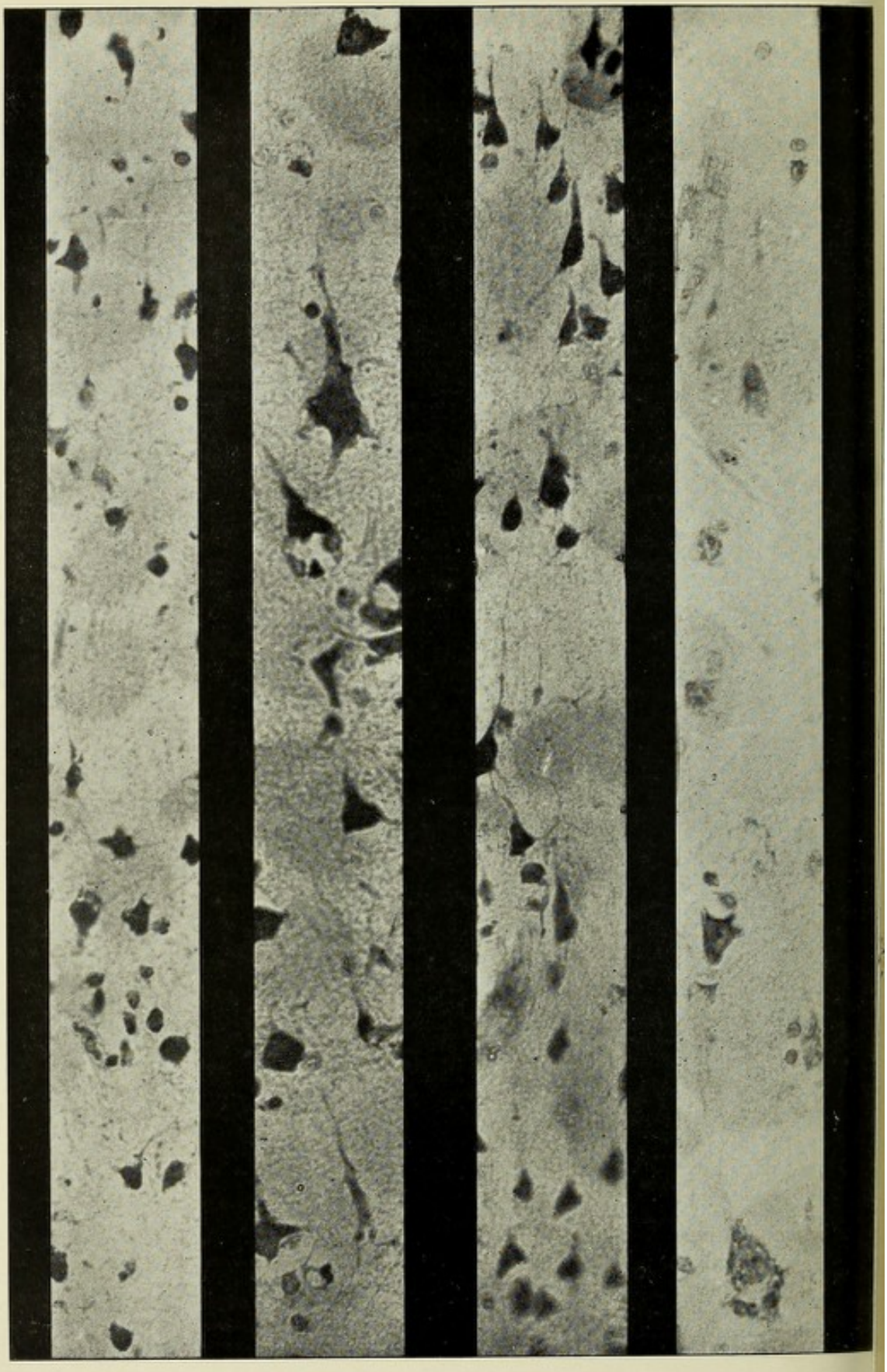
could not be obtained without reinforcement. Plantar and abdominal reflexes good. Pupils equal, 2 mm., Argyll-Robertson phenomenon. Paresis of right external rectus. Face movements good, no tremor of tongue, sphincters normal. Perimeter tracings showed considerable concentric limitation of field of vision. He attended as one of Dr. Head's out-patients until June 24, 1899. He then disappeared, and was admitted into Bexley Asylum. When I saw him he was suffering with symptoms of general paralysis. The speech was slightly slurred, the tongue tremulous, also the face muscles; there is an expression of mild exaltation, but he is able to answer questions and give a history of his occupation and illness which corresponded fairly well with the account contained in Dr. Head's notes. There is, however, some defect in his knowledge of time and place, but no pronounced delusions were discoverable. He now has some bladder trouble, diminished sensation to pain in the lower extremities; the trunk was not tested, muscular hypotonus in addition to the symptoms of ataxy previously noticed. He became progressively more demented, but rather tended to childishness than absolute loss of intelligence.

On October 12, 1900, he commenced having epileptiform seizures, and he was found by Dr. Piper convulsed on both sides and in a state of opisthotonus, his temperature was 102°, and he was sweating profusely. He was first in a comatose and afterwards in a semi-comatose condition. He had a large number of seizures of a similar character, but affecting especially the left side, and on October 15, it is noted that he had twenty-two seizures since October 12.

October 22.—It is noted that his tactile sensation was thoroughly tested, no alteration was detected, and there was certainly no hemianæsthesia present. The note further states he is now quiet, and can answer intelligently when spoken to. His memory is considerably impaired, and he often becomes confused in conversation. He passes his water naturally, and has control of his motions. No important change occurred, but progressive mental and bodily enfeeblement until February 9, when he commenced having a series of convulsions, and it is still noted that there is no hemianæsthesia.

On February 28, he commenced having attacks of vomiting, which continued for more than a week, associated with retention of urine, which persisted after the vomiting had ceased.

On March 21, the notes state "he was transferred yesterday to the general paralysis infirmary for the purposes of better classi-



(1) (2) (3) (4)

Photomicrograph of strips of the brain which are from left to right. (1) Small an medium sized pyramidal layer, top of ascending frontal, showing abolition of Meynert columns produced by destruction and distortion of the pyramids. Not a healthy cell i seen. There is a marked proliferation of glia cells. (2) The same section of the corte in the deeper layer of large pyramids. Two Betz cells are seen together, one is obviousl destroyed and has been partially devoured by phagocytes. Most of the cells ar abnormal. (3) Pyramidal layers of occipital cortex. Both as to numbers and conform ation they present a comparatively normal appearance. (4) Broca's convolution. Ver marked destruction of medium sized pyramids shown.

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fication." He is still sick at intervals; mentally he is very demented, but quite happy. He vomited at intervals up to his death on April 6.

Post-mortem examination.—Showed little in the organs of the body except in the brain and spinal cord, but it may be noted that there was an old calcareous nodule at the apex of the left lung, some emphysema of both lungs, a moderate amount of atheroma of the aorta, and considerable chronic cystitis.

Brain.—Weight of right hemisphere, 518 grammes; weight of left hemisphere, 542 grammes. Granulation of the third and fourth ventricles, dilatation of all ventricles, left lateral ventricle less than the right. Pia-arachnoid adherent in pre-frontal region mid-line, leaving erosions on stripping. External appearances of hemispheres.—Right: Obvious wasting of base of first frontal and base of inferior frontal at junction of acending frontal. Left: appears less wasted as a whole, but there is a small triangular depression due to atrophy just above the angular gyrus: appearance of gray matter of cortex is somewhat wasted, but wasting is not universal. Striation in many places indistinct. Same applies to right side.

Notes of the microscopical examination of the central nervous system.—Nissl staining of the cortex.—The small and medium sized pyramids show their processes broken off; the apical processes are either not present or cork screw. The cyto-plasm stains diffusely and imperfectly; the nucleus is often eccentric. The columnar arrangement of the cells of Meynert's layers has disappeared. The small cells of the molecular layer and the small pyramids are especially affected in the first frontal, ascending-frontal, ascending-parietal, and Broca's convolutions, but to a less degree in the occipital. The large Betz cells of the ascending frontal occur in groups, some of which appear quite normal, others show chromolytic changes, swelling of nucleus, and breaking off of processes, and some exhibit excess of yellowish pigment.

The medium and small-sized pyramids are much more affected in these regions than in the occipital lobe (*vide* Plate V.).

As a rule the vascular changes are not pronounced, but many small veins in all regions of the cortex show the perivascular lymphatics filled with basophil staining cells; there is not excessive glia cell proliferation.

Spinal cord, stained by Weigert method (each segment being examined)—Cauda equina and fifth sacral. All the posterior roots are atrophied, and almost totally denuded of fibres; some few atrophied myelinated fibres can be seen in some of the roots.

The vessels are congested, but the walls are not thickened, and they are obviously distended with blood merely from *ante-mortem* stasis, for the anterior roots, which are quite normal, contain vessels similarly gorged with blood. Third sacral and roots.—Roots same as above. Posterior column sclerosis, and almost complete denudation of fibres, except in area corresponding to the anterior two-thirds of the portion of the posterior column surrounding the posterior median fissure; behind this up to the periphery there are scattered fibres corresponding, doubtless, to the endogenous fibres belonging to the oval area of Flechsig. Second sacral and roots.—Posterior roots markedly degenerated, but contain some fibres (large) undegenerated. The whole posterior column is partly denuded of fibres, except the median oval area and the cornu commissural zone. The degeneration is most marked in the root zone, and in the zone between the two undegenerated endogenous tracts (fibres of Goll's column). Crossed pyramidal tracts, both sides degenerated. First sacral.—The roots are entirely devoid of fibres, and the whole of the posterior column is sclerosed and denuded of fibres. The cornu commissural zone and oval area are the only tracts which contain fibres, and here they are relatively abundant. Fifth lumbar.—Posterior roots not nearly so much affected as first sacral, roughly speaking, quite one-third of the fibres still left. Degeneration in the same regions as the last, but a proportional increase to the numbers in the roots of fibres entering the posterior columns. Fourth lumbar.—Ditto, with regard to fibres in roots, except the two endogenous tracts are being separated by the degenerated areas corresponding to a tract which intervenes and approximates at the middle line, separating thus the fibres of the oval area, which are pushed backwards, although there are still a number of fibres scattered in the degenerated area. The crossed pyramidal tract is seen to be more degenerated in the left than the right side; the posterior column degeneration is about equal on the two sides, and in the median line extends forwards to the posterior commissure. Third lumbar.—Ditto, atrophy of fibres of the posterior roots, otherwise the same as fourth. Second lumbar.—Ditto. Here is seen commencement of Clarke's column, and there is some atrophy of the entering roots and of the fine plexus, but it is not marked. There is less atrophy of fibres of the posterior roots. First lumbar.—Ditto, but the atrophy of the feltwort of fibres around the cells of Clarke's column is very evident as the column is more developed; the encircling tracts of endogenous fibres are, however, relatively abundant. Twelfth

dorsal.—Roots much smaller, less number of degenerated fibres, yet very marked atrophy of fibres entering Clarke's column, and of the plexus of fibres; most marked degeneration in root zone and area extending thence to mid line; slight degeneration of Lissauer's tract, moderate of postero-external triangle. Eleventh dorsal.—Posterior roots much less affected. Less atrophy of root zone and of fibres entering Clarke's column, also of other short tracts, *e.g.*, Lissauer's. Tenth dorsal.—Ditto; attached roots show very little degeneration. Ninth dorsal.—Roots of one side almost denuded of fibres, and the postero-external column and posterior horn of the same side considerably shrunken. Eighth dorsal.—Ditto. The crossed pyramidal sclerosis is becoming less obvious, and above this level is no longer recognisable by the naked eye, although microscopically a little diffuse sclerosis can be found on one side as high as the sixth dorsal. Seventh dorsal.—The posterior roots are partially but equally affected on both sides. Lissauer's tracts are very little affected at this level. Sixth dorsal.—The posterior roots are much more affected on the right side than the left. Fifth dorsal.—Roots completely atrophied both sides; both coarse and fine entering-fibres disappeared. Fourth dorsal.—Ditto. Third dorsal.—Roots atrophied both sides, one side more completely. Second dorsal.—Ditto. First dorsal.—The roots now contain a considerable number of healthy fibres, and the cornu radicular zone and the entering zone of Lissauer show very little sclerosis; still there is a great amount of atrophic sclerosis in the posterior columns owing to the atrophy of long and medium length fibres proceeding from lower levels, and although there is no posterior root degeneration above this level, it is not until we reach the seventh cervical segment that the sclerosis in the root zone entirely disappears. Eighth cervical.—Not much degeneration of root-fibres, cornu radicular and Lissauer's zones almost free of sclerosis. Seventh cervical and upwards.—The roots are healthy, and two well-defined tracts of sclerosis are seen, one overlapping the triangle of Goll's column, but a healthy strip intervening. We have seen that the roots of the lowest lumbar and sacral regions were almost devoid of fibres, and I have shown experimentally that nearly all the fibres of this triangle of Goll's column come from the fourth and fifth lumbar and first and second sacral roots, which were, correspondingly, in this case the roots especially atrophied. Above this level, up to the ninth dorsal, the roots contained a large number of healthy fibres; we must, therefore, accord to this narrow, undegenerated Λ-shaped strip an origin from these roots.

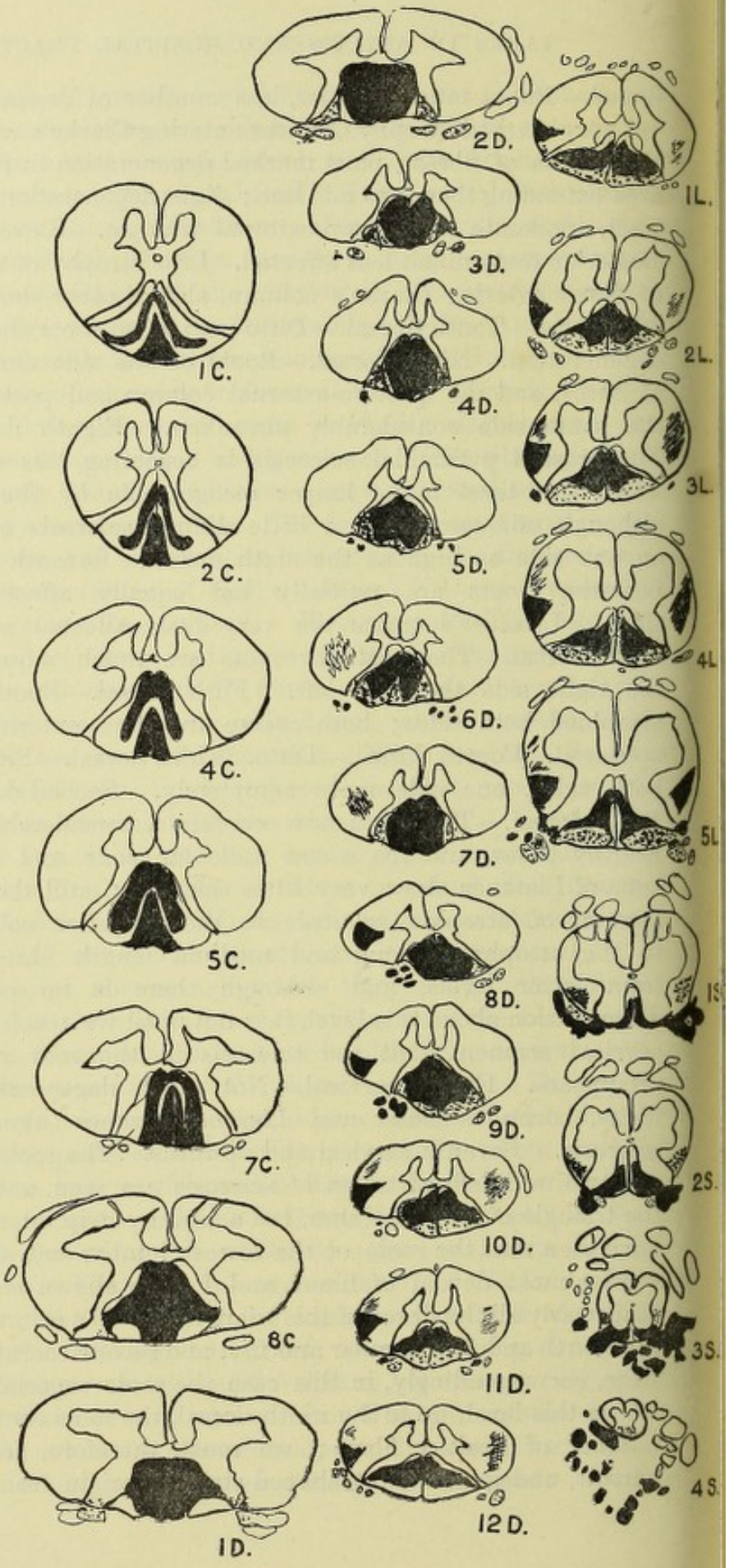

FIG. 35.

Drawing made with Edinger apparatus. Black part indicates complete or very extensive degenerative sclerosis. Dots indicate partial degenerative sclerosis. It will be observed that the eighth and ninth segments show a marked degenerative atrophy of the roots and posterior column on one side. Possibly this may be associated with the gastric crises.

*The degeneration of the crossed pyramidal tracts is not so complete as the above would indicate.

and they convey, in all probability, kinæsthetic impressions from the large muscles connecting the trunk and legs and the lower part of the trunk. At the level of the second cervical we have in front of this a Λ-shaped area of degenerative sclerosis, which corresponds to the outfall of the root-fibres in the upper dorsal and lowest cervical regions; here again it is certain that these fibres belong especially to the segments of the cord conveying kinæsthetic impressions from the small muscles of the hands, viz., eighth cervical and first and second dorsal, for I have shown experimentally that the greater part of the fibres which form this portion of the cerebro-petal kinæsthetic tract come from the eighth cervical and first dorsal segments, which we know innervate the small muscles of the hand.

In front of this at the level of the second cervical, the healthy fibres of the roots entering the cord in the cervical region, and on their way to the postero-external nucleus (Burdach's) have taken up their position in the form of a Λ, the limbs of which at their base are continuous with the root zone.

The Λ-shaped strips of fibres coming from fibres of the posterior roots are segmental, each root sending a certain number of fibres which take up a position lapping over the last, and, as we have seen, the number of fibres which each root provides is distinctly proportional to the complexity and variety of movement of the structures innervated, thus the great bulk of these fibres, viz., the fourth and fifth lumbar and the first and second sacral which innervate the muscles of the feet, the eighth cervical and first and second dorsal, which innervate the small muscles of the hand come from the mobile apices of the limbs. Sherrington has shown that from above downwards in the cerebral cortex there is spinal segmentation of the efferent path to the muscles in the motor area of the ascending frontal. Here in the posterior column we have a spinal segmenation of the afferent kinæsthetic path, and just as the greater part of the upper two-thirds of the motor area is concerned with the complex movements of the hand and foot, so the greater part of the kinæsthetic path to the cerebral cortex is concerned with conveying impressions from the hand and foot.

Association of morbid anatomical conditions with symptoms.—(1) The advanced and unusually pronounced degeneration of the lowest sacral roots—early impotence. (2) The presence of fibres in the posterior roots of the third and fourth lumbar segments in addition to the crossed pyramidal degeneration on both sides, but more marked on one side—the knee-jerks present on both sides,

but greater on the right than the left. (3) The obvious atrophy of the posterior horn in the seventh and eighth segments on one side with shrinking of the cord (as in Case 31)—the existence of gastric crises. (4) Atrophy of the tangential, supraradial fibres and molecular layer, small and medium sized pyramids, especially of the frontal and fronto-central regions—progressive dementia. (5) Slight vascular changes, little glia cell proliferation—no pronounced maniacal symptoms or delusions.

Case 49.—*Tabic amyotrophy. Tabes affecting especially the arms followed by muscular wasting of small muscles of hands. Seven years' duration, commenced with optic atrophy and terminated with mental symptoms and convulsions. Post-mortem examination.—Wasting of hemispheres, especially the right, granular ventricles, other naked eye and microscopic characteristics of general paralysis. The cord exhibited the usual appearances of advanced tabes, but also atrophy of anterior horn cells in lower cervical and upper dorsal regions, also atrophy of cells of Clarke's column with degeneration of ventral and dorsal cerebellar tracts.*

A. J., aged 42, single. Admitted to Marylebone Infirmary, May 1, 1894. For the last thirteen years, parcel-post sorter, previously a soldier. History of a buboe ten years previously. Fifteen months ago eyesight began to fail, drooping of the left eyelid, double vision, and things appeared smaller than natural. He also suffered with attacks of giddiness, lightning pains, and later weakness in the legs which prevented him walking.

The following notes were taken on admission.—Left eye, external strabismus, pupil half the size of that of right, can only just appreciate light with this eye. Right-eye movements to right made with difficulty. He cannot distinguish objects on the right side of him. Pupil much dilated. Nystagmus in both eyes in upward and downward direction. Both pupils are said to react to light and accommodation. The arms.—No apparent wasting, muscular power good, great loss of coordination in both arms, deep reflexes cannot be obtained ; complains of numbness in hands, but sensibility to touch seems normal, sensibility to heat and cold also normal. Legs.—Muscles feel flabby, patient says his legs are much thinner than they were. Knee-jerks absent; sensation normal. On trying to walk patient staggers helplessly. Taste.—Can distinguish between hot and cold, salt and sweet, but is very slow in distinguishing flavours. Bladder normal, no incontinence. Speech natural. September 6, 1894. Seen by Dr. Beevor who

found muscular sense very much impaired. Sensation to touch indefinite. No loss of tactile sensation in legs. Muscular sense much impaired. Wasting and loss of power in thumb muscles. November 19, 1895. Small muscles of the left hand give no reaction to faradic current. Small muscles of the right hand thenar, hypothenar and interossei require a stronger current than normal to give the faradic reaction. Sensation to touch is blunted on chest and arms, no loss of sensation to heat and cold. On moving the fingers the patient can tell which fingers are moved, but is unable to state what the movement is. Incomplete loss of muscular sense. Eyes.—Marked nasal hemianopsy still present. Right pupil does not react to light or accommodation, the left reacts to accommodation, but not to light. Loss of sensation to touch in both legs, but no analgesia over legs.

1899.—Examined by Dr. Beevor. Partial anæsthesia found in legs, localisation very poor. Very little tremor in the facial muscles. Patient now has power in the biceps and deltoid muscles of the left arm, and can flex and extend fingers. He has periodic attacks of exaltation.

May, 1900.—Analgesia in both upper extremities, anæsthesia on both sides up to clavicle, including arm.

On November 6, 1900, he was first seen by me, and I made the following notes. He is now semi-delirious, and not in a fit state to test sensation. On Sunday last, November 4, he had a fit, cried out, and lost consciousness for two minutes. A good deal of twitching, convulsive in character, took place on the left side, arm, leg, and face, and this lasted for twenty-four hours. He has not lost consciousness since then. Previous to this attack patient was noisy and sullen, but had no delusions. Since the attack he has seen people coming into the room and called out to them saying they were robbers. He has not refused food since fit, or had delusions about same. Since Sunday night he has suffered with vomiting and reaching and also hæmatemesis. Patient has no personal illusions. Nearly blind, right pupil 5 mm., left 4·5 mm. Inactive to light. Slight ptosis, more marked on right side, and partial immobility of right globe. He cannot be made to look when testing vision, being nearly blind in left eye, seeing best with right eye. Notes state that there is optic atrophy and hemiopia. Appetite has been ravenous. There is a little apparent loss of expression on the right side of face. Tongue very tremulous, characteristic of general paralysis, but no tremor of lips, or any speech affection. His lower extremities are very wasted, especially the dorsal flexor group of muscles of the front of the legs, both

the peroneal group, as well as the tibialis anticus and long extensors of the toes; there is talipes equino-varus in consequence, and the toes are plantar flexed; he does move legs, according to the nurse, but only in an incoordinate manner. There is marked wasting of the interrossei and muscles of thenar and hypothenar eminences, especially of the left hand. He moves right hand but not fingers much more than the left, but then only in a most incoordinate manner. He smells ether but not peppermint. He was not in a fit mental state to test sensibility, but apparently he felt pricking with a needle on the legs, as he swore violently.

The patient died January, 1901, and by the kindness of Dr. Lunn the brain and spinal cord and many of the peripheral nerves were forwarded to me for examination. Unfortunately the cord was somewhat damaged in several regions, so that an absolutely complete examination of this interesting material could not be made.

Brain.—Pia-arachnoid thickening, especially over frontal and central convolutions. Right hemisphere smaller than left, and more atrophied. Marked dilatation of lateral ventricle. Ependyma granular, marked granulation of ependyma of fourth ventricle.

Microscopical examination (Marchi method).—Left Broca, no recent degeneration. Right ascending frontal and parietal and base of second frontal, a number of recent degenerated radial fibres, especially in the two former gyri. Sections of the same convolutions at the level of the base of the first frontal showed not nearly so many degenerated fibres. Left hemisphere, results similar. Orbital lobe and olfactory nerves showed no recent degeneration.

Marchi, Pal, and Wolter's methods.—The same convolutions were examined. First and second frontal.—Tangential and supraradial fibres absent; interradial diminished. Ascending frontal.—Tangential and supraradial fibres partly diminished; interradial diminished. Ascending parietal.—Tangential and supraradial absent; interradial diminished. Broca.—Tangential and supraradial greatly diminished. First temporal.—More tangential and supraradial fibres than in other regions examined. Orbital.—Superficial fibres are diminished. Both optic nerves and tracts show very complete atrophic degeneration as far as geniculate bodies.

Nissl method.—All the above regions of the cortex were examined, and showed marked congestion of vessels, and plasma cells in perivascular lymphatic sheaths. The cells of the deeper layers of the cortex present a fairly normal appear-

PLATE VI.

FIG. 2.

FIG. 4.

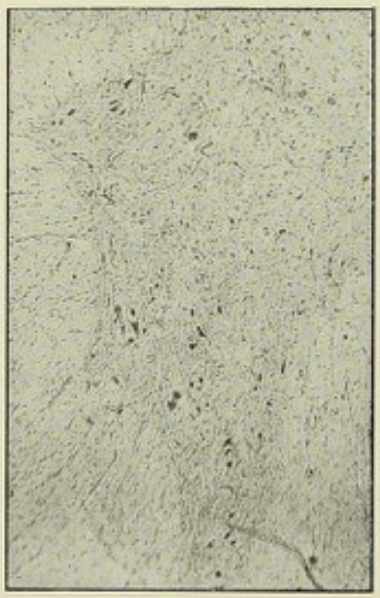

FIG. 1.

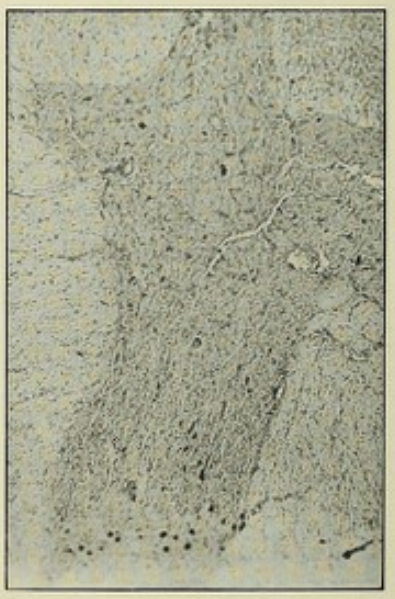

FIG. 3.

TABES IN ASYLUM AND HOSPITAL PRACTICE.

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ance, in spite of the marked vascular changes. The outline of these cells is well defined, and their apical processes straight. The columns of Meynert are fairly well defined. The nearer the surface is approached the more numerous are the atrophied and altered cells, processes being broken off and no longer retaining a pyramidal form, staining imperfectly and showing changes both in the nucleoplasm and cytoplasm. The existence of a cell is often only determinable by the persistent stainable nucleolus. The glia cell proliferation is slight in proportion to the vascular congestion and the cellular decay and destruction. The vascular changes are as marked in the deeper layers as in the superficial; the cell proliferation in the perivascular sheaths is mainly due to lymphocytes. The vascular and cell changes were much less obvious in the calcarine region. It was concluded that the cell change in the superficial layers was for the most part primary and chronic and independent of the vascular, which was acute, and coincident with the attack of acute mania which had supervened a few months prior to death.

Spinal cord.—(Nissl method). Sixth, seventh, eighth cervical, first, second, and fourth and tenth dorsal, first and fifth lumbar and first and second sacral examined. Sixth cervical.—No atrophy of anterior horn cells. Seventh cervical.—Partial atrophy of the anterior and internal groups of anterior cornual cells, the lateral groups being normal, or nearly so. Eighth cervical.—There is marked atrophy and disappearance of the ganglion cells of the anterior horn of the left side, and on the right side the cells are greatly diminished in numbers. Many of them have but few processes, the Nissl-bodies are absent or diminished, their sides convex instead of concave, greatly diminished in size, and in many instances the cytoplasm appears to be filled with yellow pigment granules. First and second dorsal:—There is hardly a ganglion cell to be seen on either side in the anterior horns. On the right there are a few cells in the lateral horn, but these are mostly degenerated. Not only is there a disappearance of the anterior horn cells, but also of the more numerous smaller cordonal cells of the gray matter. There is not a great excess of glia cells but large, thin-walled, dilated veins due to the distension of pre-existing vessels were observed, but no old or recent hæmorrhages.

In the lumbo-sacral region the anterior horn cells show chronic atrophic degeneration similar to, but less advanced than that in the cervico-dorsal region. There is also atrophy of the cordonal cells of the gray matter at the base of the anterior horn and throughout the posterior horn.

Sections of cervical, dorsal, and lumbo-sacral regions stained by Marchi method exhibited scattered degeneration of recent character in the antero-lateral tracts, especially the crossed pyramidal; also a little in the posterior columns in regions corresponding to the cornu commissural zone, where practically fibres only exist.

Examination of the spinal cord by Weigert and other methods exhibited a marked degenerative atrophy of all the posterior roots and the exogenous fibres of the posterior columns of the spinal cord, without any marked vascular change to account for the same. The membranes were not much thickened. A considerable substitutive overgrowth of glia tissue exists in the posterior columns, so that they are not so much shrunken as is often the case. Shrinkage of the spinal cord generally to the size of that of a child in the first and second dorsal, fifth lumbar and first sacral regions, atrophy of the root zone in these regions and of the anterior roots was observed. With the exception of a few fibres in the cornu-commissural zone close up to the posterior commissure (*vide* Plate VII.), the whole of the endogenous system of fibres are destroyed. Probably this may be associated with the destruction of the cordonal cells above noted. There is a marked atrophy of the fibre plexus of the column of Clarke, and an atrophy of the cells at all levels which accounts for the partial atrophic sclerosis of the ventral and dorsal cerebellar tracts, especially the former. There is also at all levels an atrophy of the cells of the posterior horn accounting for the disappearance of all the descending endogenous fibres and a great part of the ascending (*vide* Plate VII.). There is marked flattening and shrinking of the cord in the cervico-dorsal region; the posterior columns have in this situation, but nowhere else, a concave instead of a convex external border, indicating long-standing atrophy and shrinkage.

A considerable portion of the dorsal spinal cord had been damaged in its removal, and therefore was not of much use for examination, but in the light of Purves Stewart's case of crush of the cord in the lower cervical region, with *descending degeneration* in the comma tract, continuance of the same into the posterior external triangle, septo-marginal, oval area, and median triangle of Phillipe (all the fibres of which are completely absent in this cord), and of the fact that there is definite evidence of shrinkage of gray matter and disappearance of cordonal cells in the lumbo-sacral and cervico-dorsal regions, the following facts—(1) atrophy of small muscles of hand and foot, (2)

PLATE VII.

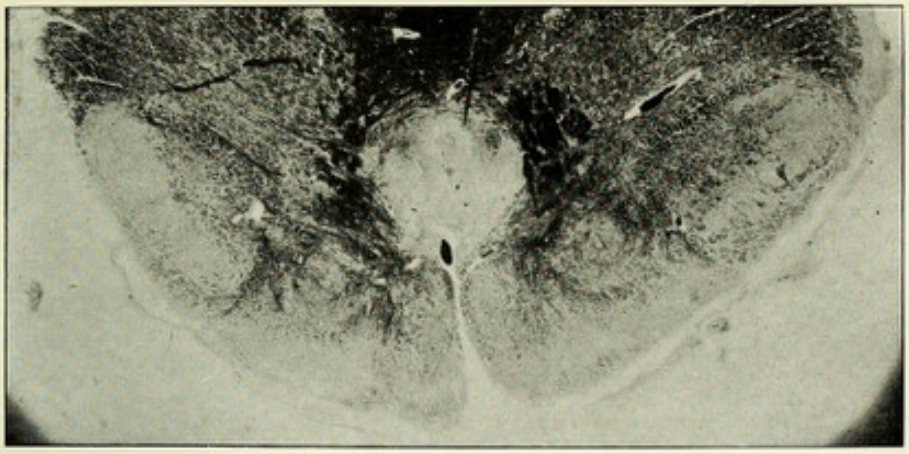

FIG. 1.

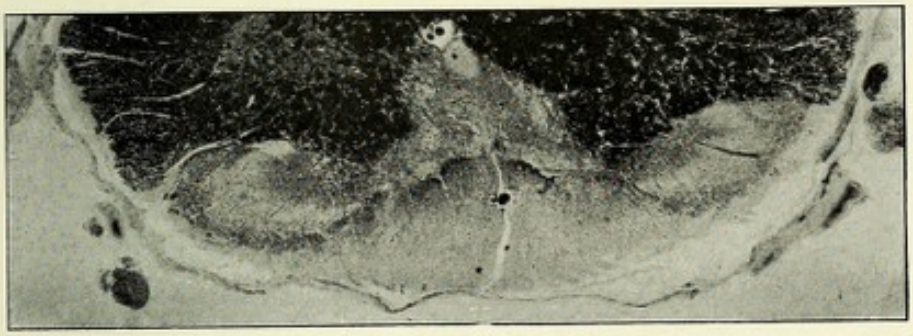

FIG. 2.

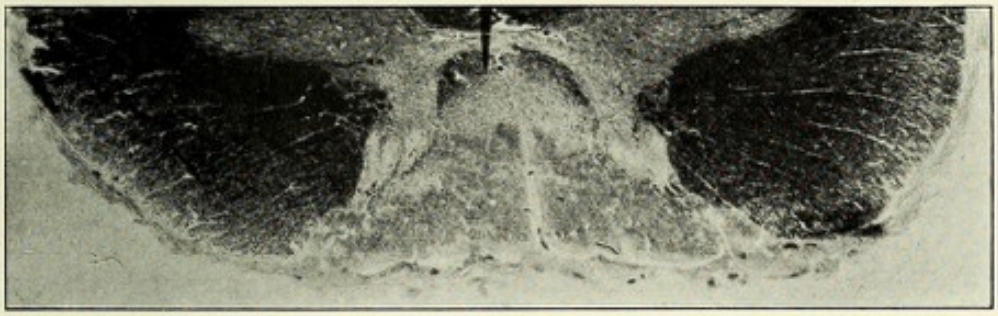

FIG. 3.

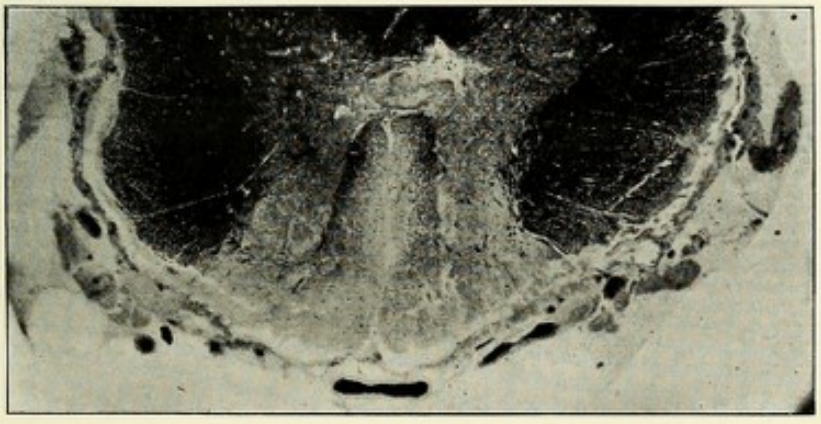

FIG. 4.

TABES IN ASYLUM AND HOSPITAL PRACTICE.

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atrophy of anterior horn cells in cervico-dorsal and lumbo-sacral regions, (3) atrophy of gray matter and cordonal cells in the region of outflow of nerves to hand and foot, (4) atrophy of endogenous systems of cordonal cells and intercalary neurons—show reason for the argument that not until there is atrophy of the intercalary neurons and of the cordonal cells, which give off fibres associating different segments, will the anterior horn cells be deprived entirely of stimulus, and, therefore, undergo regressive atrophy. Destruction of the exogenous systems alone will not suffice, as I have found by cutting a number of posterior roots.

In the lumbo-sacral region, where the muscular atrophy was not so advanced, nor the cell atrophy of the anterior horns, the primary nonvascular origin of the process was evident.

I look upon the morbid processes which occurred in this extreme case in the following order: (1) Degeneration of posterior spinal afferent neurons. (2) Degeneration of their association neurons. (3) Degeneration of intercalary neurons, resulting in cutting off completely the stimulus from anterior horn cells, for Schäfer has shown experimentally that Von Monakow was correct in his assumption that the crossed pyramidal fibres probably do not directly arborize with the anterior horn cells. (4) Regressive atrophy of the anterior horn cells now deprived of all stimulus, and as a result of this, degeneration of the motor fibres and muscular wasting. Slight degeneration was found also in the pyramids of the medulla, and the crossed and direct pyramidal tracts in the spinal cord. Dr. Purves Stewart has advanced as an argument against the neurone theory the fact that in his case of crush of the cervical cord he has found degeneration in the internal arciform fibres, and, therefore, the degeneration has not been confined to one neuronic system. This to my mind is full of fallacy, for such a severe lesion as a crush of the cord might well give rise to signs of Marchi degeneration (far removed from the seat of the lesion) from vascular disturbances and other causes. I have had considerable experience in examining the medulla after section of the posterior roots, and I have never observed, even after cutting all the posterior roots of one side supplying the upper limb with sensation, any degeneration in the internal arciform fibres and fillet. I have examined now the cords of twenty-eight cases of tabes and tabo-paralysis, and even in such extreme posterior column degeneration as this case presents, I have never seen any atrophy of the internal arciform fibres, although there is complete fibre plexus atrophy

of Goll and Burdach's nuclei. In refutation of this statement of Purves Stewart I call the attention of my readers to Plate VII., fig. 1, showing the condition of the internal arciform fibres as they leave the posterior column nuclei to decussate in the mid line to form the fillet.

Another fact worthy of note in this case is the existence of some fine fibres in the roots of the cauda equina, and also the entering rootlets of the lumbo-sacral spinal segments. These fibres belong to the external group of fibres, and break up immediately into fine collaterals around the neurons of the substantia gelatinosa. According to Bechterew they subserve cutaneous sensibility. When the sections of the spinal cord were stained by *Bolton's iron alum method* these fibres were seen fairly numerous in some sections, whereas the inner group of coarse fibres are entirely absent in the roots and in the cornu radicular zone. We can associate this anatomical finding with the fact that the patient was apparently aware of rough pricking of his legs when I tested him, although he was completely helpless and almost blind.

The ulnar nerves hardly contained any fibres, the nerve was shrunken, and the fasciculi consisted mainly of thickened epi-peri and endoneurion. The median was only slightly affected, and the musculo-spiral not at all. The sciatic nerve was, like the other nerves, much smaller than natural ; microscopically all the bundles showed a marked outfall of fibres, but the atrophy was not nearly so marked as in the case of the ulnar nerve.

This quite agrees with the clinical history of the affection commencing in the arms, and of the paralysis affecting the small muscles of the hands.

Case 50.—*Tabo-paralysis, ataxy of some standing. Attack of mania, grandiose delusions and progressive dementia, death six months later.*

J. H., occupation bedstead packer, using a treadle all day long, admitted to Cane Hill, September 20, 1901, aged 39, from Shoreditch Infirmary. No family history of insanity or nervous disease.

Physical condition.—Irides blue, pupils in faint light, right $3\frac{1}{2}$, left $2\frac{2}{3}$, accommodate to $2\frac{1}{2}$ and 2 mm., strain accommodation, equal, 2 mm. No reaction to light, both somewhat irregular, especially the right. Extremely marked tongue and jaw jerk, patient cannot satisfactorily protrude his tongue owing to the jerk.

Speech very slow and drawling, with considerable slurring, and at times such marked hesitation that it is impossible to make out what he wishes to say. Marked tremor of lips. Left upper abdominal reflex absent, the others normal. Scar of a stab to the left of the xyphoid cartilage and a hole through into the abdomen which readily admits the finger. Patient says that it was done by his wife with a butcher's knife. Sensation is delayed (both touch and pain) over the abdomen (which is retracted) and chest to about two inches above the nipples, and on the front of the chest up to this level there seems to be a condition almost of analgesia. The results are, however, unreliable owing to the mental condition of the patient. The heart is normal. There are signs of old consolidation of the apex of the left lung. There are several papery scars on the legs, one of which above and inside the left knee is markedly serpiginous in outline. The knee-jerks are absent, patient feels touch and pain, but has no true plantar reflex. If either touched or pricked on the soles of his feet, he, after a considerable latent interval, rapidly withdraws the leg, wriggles in bed, and complains bitterly of being hurt. Hypotonus to 15° beyond a right angle, arm reflexes absent, right grasp fair, left grasp very strong. Patient cannot support himself on his legs without help. On trying to walk, there are extremely marked ataxic movements of the legs, with stamping of the heels; Romberg sign is well marked. There is a marked scar (patient says of a "chancre") on the left side of the corona on the under surface of the foreskin. Patient volunteers the information that he has had "syphilis."

General condition.—Patient is a very feeble and emaciated man, who is unable to walk or attend to his own wants. He is at present somewhat drowsy owing to his having had a powder administered yesterday evening. This was given owing to his extreme restlessness, lest he should be injured by throwing himself about. During examination patient either replies drowsily to questions or is restless and groans. He complains of pains in his "body, and legs, and all over." No more definite information can be obtained. He looks very ill, and his face is drawn and pinched, but otherwise expressionless.

Mental state.—Patient is confused, very self-satisfied, his statements appear to be altogether unreliable; he occasionally replies to questions, does as he is told, or notices what goes on around him. He says he is married and that his age is 29. He has "a lovely wife and a beautiful daughter, he has been married sixteen years, he has nineteen daughters, he can work the typewriter and is a

printer, he was a sailor and in the Navy." When left to himself he rambles on about going to Australia, to diamond mines and rambling about the coast.

The discs are normal. They are both very pale and suggest moderate atrophy at first sight. Patient can, however, read easily with either eye at about the normal distance, and, consequently, the pallor is normal.

October 10.—The patient's mind is somewhat clearer, and he gave me the following account of himself. He had, in 1884, a sore followed by secondary symptoms for which he was treated some months with mercury pills. He was aged 20 at the time. A few years later he became acquainted with a woman who had left her husband, and they had lived together as man and wife up till six months ago, when she deserted him, and to this he attributed his mental affection. The history he gave, however, suggested that she had been compelled to leave him on account of his habits and infirmity. His illness began three and a half years ago with pains in the legs, difficulty with the bladder and bowels, followed by ataxy and ocular paralysis. He went to King's College Hospital and was admitted under Dr. Ferrier. Soon after this he seems to have had an apoplectiform seizure and was taken up by the police. He has had loss of sexual power for three years ; this was preceded by satyriasis. The woman he lived with bore him no children.

Physical condition.—Knee and triceps jerks absent, plantar and cremasteric reflexes also absent. Joint sensation in toes lost, not however in ankle. Joint sensation in fingers lost, ataxic gait, inability to touch nose with eyes shut, hypotonus to a right angle. No anæsthesia of the trunk or limbs detected and no analgesia. This is fairly reliable. He has very marked tremor of the lips and tongue, and the speech is very slurred. The smell is defective, as he is unable to recognise the strong odours of assafœtida, cloves, or peppermint. His taste is good, recognises quinine, sugar, and acids. Pupils remain the same as when last examined. He is able to walk, and he volunteers the remark that he was much worse in this respect at one time.

Mental condition.—His knowledge of time and place and memory are fairly good, but he has exalted and grandiose ideas about obtaining watches and jewels, and making large sums of money by pawning them. The attendant states that he is better and has control over his sphincters.

March 1.—Death—Autopsy refused.

Case 51.—*Ataxy of some years duration in a man aged* 39. *Onset of cerebral affection manifested by epileptiform seizures, mania, with grandiose delusions. Death eighteen months after onset of mental symptoms. Marked cerebral lesions characteristic of general paralysis, and marked spinal lesion characteristic of tabes dorsalis.*

R. S., aged 39, was admitted to Cane Hill suffering with mania and grandiose delusions. The history was that he had suffered for eighteen months from locomotor ataxy, for which he had been a patient at the Croydon Hospital. The supposed cause of his mental affliction was worry and family troubles occasioned by his loss of occupation as a gardener. He had been married eleven years, and had a family of five children. No history of syphilis was obtainable, and there were no signs on the body.

Physical condition.—Nutrition poor, muscles wasted; he has marked locomotor ataxy. The pupils small, equal, inactive to light, react to accommodation. Knee-jerks absent; gait, walks with a wide base, and is somewhat unsteady. Speech markedly tremulous.

Mental state.—Conversation rambling, irrational, and incoherent. Says his brothers are coming up to the Croydon Flower Show, that they will stay three months; that he has four elephants and fifteen horses.

After two or three months in the asylum his ataxy considerably improved. From time to time he had convulsive seizures, from which he recovered, but his mental condition became progressively worse, although sometimes after the fits had passed over, he would apparently be somewhat brighter. He died of exhaustion of general paralysis eighteen months after admission. (Abstract of notes by Dr. Donaldson.)

Summary of post-mortem notes.—Membranes thickened, opalescent, and adherent to the brain cortex; excess of cerebro-spinal fluid, all the ventricles somewhat dilated and markedly granular. Left hemisphere weighed 20 grammes less than the right.

Summary of results of microscopical examination: Brain.—Portions of the prefrontal, Broca's, and central convolutions examined by Nissl and Pal methods showed the characteristic changes of a fairly advanced case of general paralysis, viz., vascular engorgement, thickening of membranes, proliferation of glia cells, dilatation of perivascular lymphatics, with cellular proliferation. Disarrangement of Meynert's columns owing to

acute and chronic destructive changes in the cortical cells, especially of the molecular layer and the small and medium sized pyramids. Atrophy and partial disappearance of the tangential and supraradial fibres, which is most marked in the prefrontal and Broca's convolutions.

Spinal cord.—All regions examined, and showed a naked eye disappearance (with glia substitution) of the intramedullary projections of the root fibres in the lower cervical, dorsal, and lumbo-sacral regions. The endogenous systems of fibres were in this case but little affected. The cornu commissural tract possessed the normal wealth of fibres ; there may have been some sclerosis in the comma tracts in the mid-dorsal region, but there was an abundance of fibres in the posterior internal zone, and these could be traced to the lower dorsal region as a strip of fibres amid the sclerosed tissue extending along the periphery of the cord, and at a lower level massing into a small triangle, which, again, at a still lower level, could be followed as the septo-marginal ending in the oval area in the lower lumbar region, and finally continuous with the median sacral triangle.

GROUP 4.—Tabo-paralysis with Marked Speech Affection.

Case 52.—Tabo-paralysis, five years' duration from onset of symptoms, history of hereditary insanity in mother and brother. All the family in the publican line and given to drink. The disease commenced with fits, speech affection, alternate excitement and depression, grandiose and sexual delusions and perversions. Considerable ataxy without anæsthesia, or loss of joint sensation. Superficial and deep reflexes exaggerated. Death from dysentery. Typical general paralytic brain, direct and crossed pyramidal degeneration of spinal cord and typical posterior column degeneration of ataxy. No degeneration of posterior roots or Lissauer's tract of fine fibres; only coarse fibres of entry going to cornu radicular zone. Cord very small.

B. C. A., aged 50. Admitted to Colney Hatch, May 29, 1900. Occupation for fifteen years barman and cellarman, then barber for six years, and finally for the last seven years willing to take any odd job such as painting and decorating. From the account he gives me he evidently lived a very racketty life, drank hard and indulged in marked sexual excess. He denies venereal infection, but there is a scar on the right groin, enlarged inguinal glands, and several papery scars on the legs. He says he has three children, the first of whom was born five years after marriage; two twins had

previously died. It was ascertained that his brother Walter had been in the asylum fifteen years, suffering with delusions of persecution, attempted suicide—cause drink and heredity. Mother died in asylum forty years ago, went out of her mind on account of worry brought on by her husband's gambling, drinking, and immorality. For two months prior to admission to the asylum, he had been having fits. This agrees with the statement of his wife in the Case Book. Prior to admission the wife states he was at times violent, at other times depressed. She and her children were afraid of him when he was violent. She said that he had similar fits some years before admission; he had recently become intemperate and given way to gambling. She had noticed an alteration in his speech two years before admission.

Physical condition.—He has an ataxic walk, heels down first, wide base and considerable incoordination. He has no cord-like constriction, he has no difficulty with his water or bowels. Knee-jerks brisk on both sides, muscles well-developed and strength good. There is marked hypotonus of the hamstring muscles both sides and incoordination in the legs, thus he could not place right heel on his left foot or knee, with his eyes shut. No loss of sense of position of joints, and no anæsthesia of the skin could be detected, nor was there any delay in response. Superficial reflexes all markedly increased. Romberg's symptom very slight. Speech very slurred and indistinct. According to the attendant it has become progressively worse. Tongue tremulous and very jerky on protrusion, marked tremor of the lips. Pupils pinpoint, right 2 mm., left a shade less. Inactive to light, react to accommodation. Roughly tested colour vision normal, no limitation of the visual field.

Mental condition.—He principally exhibits exaltation, delusions, and mild progressive dementia. Expression is rather vacant, but is easily excited to mirth especially when relating his love affairs and virility (sexual delusions and perversions). Orientation time and place fairly good. His memory he says was fairly good. He knew correctly that his wife came to see him last Sunday with his eldest son. He seems to give a rational account of his two sons' employment. He gives correct answers when asked to multiply three by four and six by seven; still answers correctly when the figures are reversed. He remembers songs and words well. Mentally he is not much demented considering the very marked speech affection. He has had no fits since he has been in, there is more difficulty in articulation than anything else. He reiterates that too many women is the cause of his illness. He

thinks that if he puts camomile poultices over himself, he would be quite well.

Death on January 28 of acute dysentery.

The brain showed very marked wasting of fronto-central regions, thickening of all the membranes and atrophy of the convolutions everywhere to some degree, but especially in the frontal and central regions. Cortex diminished, striation very indistinct. Ventricles dilated, ependyma granular. Naked eye appearances typical of general paralysis. There was a naked eye change in the posterior and to a less degree in the lateral columns of the spinal cord which could be fairly accurately localised after hardening in Müller's fluid. The degenerative atrophy occupied the root zone; throughout the cord it could be seen distinctly entering Clarke's column, and in the upper dorsal and cervical regions there was obvious degeneration of Goll's column. Further particulars are given in the complete microscopic examination.

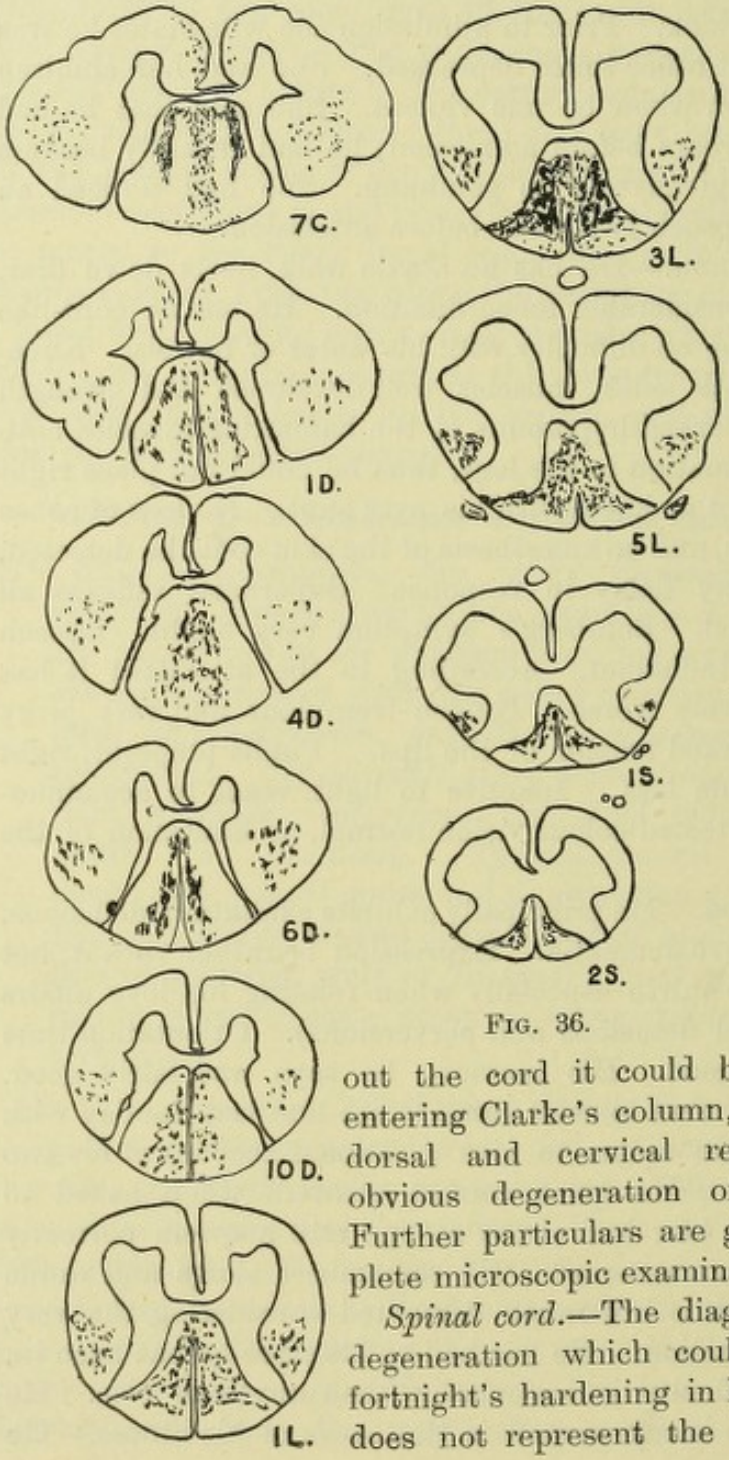

FIG. 36.

Spinal cord.—The diagram indicates the degeneration which could be seen after a fortnight's hardening in Muller's fluid. It does not represent the actual degenera-

tion which was found by Marchi method, which I will now briefly describe.

Lowest cervical and upper dorsal.—Recent degeneration in crossed and direct pyramidal tracts, degeneration in root zone, some slight degeneration in comma tract.

Mid dorsal.—Very marked degeneration in root zone, in cornu radicular zone, both crossed pyramidal tracts and comma tracts. The same applies to the lower dorsal. At the base of Clarke's column are seen a number of fine degenerated fibres (collaterals), proceeding from situation of crossed pyramidal tract.

Lumbo-sacral.—Marked degeneration in cornu radicular zone, root zone, septo-marginal and oval area, very numerous fine black particles in the pyramidal tracts of both sides, and a few, but not nearly so many coarse fibres as in the sections above described. In the attached roots of not one of these segments examined (and a large number of sections were looked through) could I observe any degenerated fibres in the posterior roots by the Marchi method.

Conclusions.—It appeared, therefore, in this case at any rate, that the degeneration commenced in the intramedullary portions of the neuron. Many sections showed an attachment of the nerve roots entering the cord (*vide* photomicrograph, fig. 41, p. 274). No degeneration extramedullary, but immediately after piercing the membranes (and therefore losing their primitive sheath), the fibres are completely degenerated.

This acute degenerative process is very interesting as showing either support for Redlich and Obersteiner's Theory of Meningeal constriction, or, as I believe, an acute primary degenerative process commencing in the portion of the neuron where the primitive sheath is absent. The membranes are not thickened over the posterior columns nor at the root entry. Another point of interest is that although there was a considerable outfall of exogenous fibres in the lumbar enlargement as shown by the Weigert method of staining, yet a considerable number of fibres can be seen entering the root zone. Of course, a great number of these may be degenerated as shown by the Marchi method.

Such an amount of degeneration would, I think, under ordinary circumstances, have been sufficient to abolish the knee-jerks, but there was a crossed pyramidal degeneration both sides. There was very considerable recent degeneration of descending and ascending endogenous fibres, and this may partially account for the pronounced ataxy.

Case 53.—*Tabo-paralysis, first symptom inability to use his tools, then has left-sided fits, affection of memory and speech, development of ataxia, no cutaneous sensory or visceral symptoms. The degeneration apparently has affected the motor afferent and efferent systems, and the kinæsthetic centres of verbal and written speech. No delusions.*

J. F., aged 36, cabinet-maker, transferred from St. Pancras Workhouse Infirmary to Hanwell, December, 1901. In April, 1899, he was an outpatient at Queen Square Hospital under Dr. Beevor, who has kindly furnished me with the following notes :—Syphilis ten years before. History of two fits left-sided. Pupils R. > L. reacted to accommodation, not to light. Tremor in tongue and lips. Speech suggested general paralysis. Mentation slow, fixation bad. Romberg's symptom, ataxic gait. No lightning pains, no anæsthesia, no analgesia. Sphincters not affected. Memory very bad, nil ophthalmoscopic. Diagnosis general paralysis or tabes, with an inclination to the former.

This account closely corresponded with the account the patient gave me concerning the fits and his subjective symptoms, so that his answers may be considered reliable as to his present condition. Married seven years, no children and no miscarriages. There is a scar in the groin, cervical and inguinal glands enlarged. He states that he was treated with strychnine by Dr. Beevor, and that he had had two left-sided hemiplegic attacks affecting his speech and memory. He fell down suddenly in the Euston Road, losing his senses, and when he came to, his speech was affected and his memory bad. He states that for some time previous to the fits he was unable to do his work, because he could not use his tools.

Physical condition.—His gait is markedly ataxic. Romberg's symptom marked. He has no pains. There is incoordination in the hands and some loss of sense of position. He suffered with bladder trouble while in the workhouse, necessitating use of catheter. He has now no control over the sphincters. Never had double vision. Pupils 4 mm., irregular, A. R. Knee-jerks absent both sides, triceps and wrist tap obtained on both sides. Some incoordination in the hands. Face congested, flushed, greasy skin, emotionless, blank expression. Tongue and lips tremulous, speech hesitant and syllabic. Sensory. No analgesia or anæsthesia discovered anywhere. Superficial reflexes present.

Mental state.—Slow reaction. Thinks he is in St. Pancras Workhouse, he knows the day of the month, but there is a

moderate amount of dementia. He has no delusions, hallucinations, or illusions.

April 9, 1902.—Physical condition very much the same, except that he is more helpless. He lies with legs and thighs semi-flexed, but feet plantar flexed and inverted owing to weakness of dorsal flexors of feet and peronei. The feet are blue and cold, and the plantar reflexes are now absent. Epigastrics are present. It is impossible, owing to his mental state, to test in a reliable manner his cutaneous sensibility; but there is undoubtedly hypalgesia all over the body, and analgesia of the lower part of trunk and legs. There is paresis of left external rectus and double vision. Pupils 4 mm., equal A. R. He cannot stand without support, and if he closes his eyes he sways to and fro. His speech is very syllabic and slurred. When asked to write he was unable; he tried to hold the pencil between the middle and ring fingers, closing his fist, but he could make no movements, and dropped it. He cannot walk without assistance; the gait, when supported by an attendant, was noted. He walks with rather a wide base, brings his heels down first, throws out and circumducts advancing leg so as to clear the ground with the foot which is plantar flexed and inverted; he does not flex the knee at the commencement of the step, but keeps it extended, and the ground is cleared by abduction and circumduction of the leg. He uses his right leg more than his left for progression.

Case 54.—*Tabic paralysis, Babinski's sign, successive right-sided fits, absent knee-jerks, aphasia, hemiparesis and hemianæsthesia of a temporary character at first, but followed later by permanent defect, death in a fit, great wasting of left hemisphere, no coarse lesion, marked pyramidal degeneration, direct and crossed in the cord, with old posterior column sclerosis. Large amount of cholin in the blood.*

D. G. W., aged 45, journalist. Was admitted into Charing Cross Hospital, February 20, 1902, having fallen down in a fit. The next day when I saw him he was conscious, but unable to speak or write, nor did he seem to understand what was said to him. When talking to the students on his case, he behaved like a man who was listening to a foreign language, and who understood every now and then a word that was said. The pupils are unequal and inactive to light, active to accommodation. The knee-jerks are absent on both sides, and there are typical syphilitic scars on the legs. The plantar reflex on the right side

gives a well marked extensor response; there is some rigidity in both the right arm and leg, and hemiparesis. Slight paresis in the lower part of the face on the right side; lips tremulous, tongue tremulous, protruded very slightly to the right. Diagnosis tabic-paralysis. A few days later I ascertained from a relative that this patient had been treated for locomotor ataxy. He also informed me that the patient had for some time acted strangely. There was no history of insanity in the family. The patient in a few days partially recovered his speech. He left the hospital, had another fit, and was taken into King's College Hospital; there he became troublesome, showed signs of dementia, and was certified and transferred to Claybury Private Asylum, March 11, 1902, where I saw him again. He did not recognise me, although he was able to speak now, and to tell me that he was a journalist connected with the *Globe* newspaper. I made the following notes: There is no doubt that he is suffering from paralytic dementia with tabetic symptoms. His speech is hesitating, stumbling, and words are occasionally clipped and slurred; his comprehension is poor, and he has marked loss of memory, in consequence of which he is incoherent and rambling in his conversation. His orientation is defective. Pupils unequal, irregular, left the larger, both react very sluggishly to light. March 22, the patient gradually became hemiparetic on the right side; the arm showed entire loss of muscular sense and tactile sensibility, though general sensibility from the coarse point of view was present; he was unable to perform any voluntary movement with it, but simply made spasmodic attempts to do what was desired; he could not place a pencil in the hand owing to the extreme anæsthesia of the fingers. The grasp, however, in the right hand was very good on bilateral squeezing, although delayed, and he was also able to perform bilateral movements, such as raising both arms above the head, and putting them in various positions, especially if the action was first performed once or twice by the left hand. He occasionally got a word out, but very rarely, most of his replies being of the nature of gibberish. He, however, understood perfectly all that was said to him, and at times, when not endeavouring to use a particular phrase or word, got out a short reply quite distinctly. Right lower face paretic. These facts, together with subsidiary details not referred to, point almost conclusively to a temporary cortical vascular disorder, involving in order of severity the face, arm, and leg areas.

At the commencement of April he began to get wet occasionally, and more demented. Since the last fit he has not recovered the

power in his right arm. The mental condition became worse, and on June 13 he had another fit, became unconscious, and died suddenly in a convulsion, 3.25 a.m.

Abstract of post-mortem notes.—Cause of death, exhaustion, general paralysis. Cerebral hemispheres, right 542 grammes, left 480 grammes. Ventricles.—Dilated and granular, especially the fourth. Pia-arachnoid.—Thickened and somewhat opaque, especially over frontal and central regions of left hemisphere, which is markedly atrophied. Spinal cord.—After hardening in Müller for two days, exhibited characteristic gray degeneration of posterior columns, degeneration of both crossed pyramidal tracts and the direct tract of the left side, very obvious to the naked eye. The blood taken from this case yielded, comparatively speaking, a large amount of choline, thirty minims of blood being sufficient to show large numbers of octahedral crystals of good size of the double salt of choline and platinum. All the vital organs were, to the naked eye, perfectly healthy; there was no obvious organic disease; the body was well nourished, and no morbid appearances presented themselves, except in the above-mentioned cerebral condition.

We might, therefore, presume that the patient may have really died from toxæmia; for although choline is a comparatively feeble poison, yet with other bodies of the cholin group existing in the blood and cerebro-spinal fluid in such large amount as the above test indicates, might suffice to produce toxic effects.

Naked eye examination of brain after hardening in Formol-Müller and stripping the membranes.—On both sides there is thickening of the membranes, the left hemisphere is obviously smaller than the right, the convolutional patterns of the two hemispheres are complex, and pretty closely resemble one another in the form, number and distribution of the sulci and convolutions. Placed with the mesial surfaces in apposition, the top of the fissure of Rolando on the two sides being made to approximate, it is shown that the prefrontal tips also approximate, and there is convolutional symmetry in size, and fairly in form of the halves of the cerebrum; but behind the fissure, all the convolutions of the left hemisphere are markedly atrophied, especially the ascending parietal, the para-central lobule, the angular and supramarginal convolutions, so that the tip of the occipital lobe of the right hemisphere projects quite half an inch further back than the left—this being mainly due to atrophy of the convolutions behind the fissure of Rolando. The temporal lobe of the left hemisphere is smaller than that of the

right, and especially noticeable is the difference in size of the first temporal on the left as compared with the right side. The posterior third of this convolution is very small and atrophied. The atrophic process then has affected, especially in this journalist, the auditory and visual word centres. The appearances of the left half of his brain conforms to the symptoms manifested during life, viz.—sensory aphasia and paraphasia, confused ideation of verbal and written speech, although he could utter articulate sounds. His speech defect was not like that of an ordinary general paralytic. Preliminary microscopical examination of the brain showed characteristic changes of an acute nature, cell and fibre destruction, glia proliferation, plasma cells around the vessels. Medulla and spinal cord.—Abundant degeneration was found by the Marchi method in both pyramids of the medulla, especially the left, and in the left direct and right crossed pyramidal tracts (*vide* fig. 42). There were also numerous degenerated fibres in the right direct and left crossed pyramidal tracts. By the Weigert method there was naked eye degeneration of the left direct tract in the cervical region, and of the right crossed pyramidal tract, and to a much less degree the left throughout the cord. The crossed pyramidal and pre-pyramidal tracts on both sides stain indifferently as compared with the rest of the antero-lateral regions of the cord, as if there was some chemical change in the myelin apart from the sclerosis due to overgrowth of glia tissue. The posterior columns show a marked sclerosis corresponding to the disappearance of the intra-spinal projections of the posterior roots. There is a naked-eye sclerosis of Goll's column in the cervical region; marked atrophy of the cerebello-petal fibres of the lumbo-sacral roots, and correspondingly of the plexus of fibres around the cells of Clarke's column in the lower dorsal and upper lumbar segments; and marked atrophy of the root zone in the lumbar and sacral regions. The lower lumbar and sacral roots are considerably denuded of fibres, half to one-third. Lissauer's tract of fine fibres is apparently little or not at all affected. There is no atrophy of the endogenous systems of fibres. The septo-marginal and oval area and sacral triangle show no outfall of fibres. The symptoms and signs observed during life can be explained by the morphological changes. The spinal lesions correspond to an early condition of the first stage of ataxy in which the skin sensibility of the soles persists, hence the plantar reflexes were obtainable. On the right side there was marked sclerosis of the crossed pyramidal tract, hence the extensor response in the sole reflex. Although

there was this marked pyramidal degeneration, the knee-jerk did not return, because of the extensive degeneration in the root zone breaking completely the reflex path of spinal muscular tonus.

Case 55.—*Tabetic general paralysis, in which noticeable symptoms were first mental, and subsequently spinal. No history of insanity in the family. Analgesia all over the body.*

H. E., aged 38, admitted to Hanwell, January 29, 1896, occupation carman, married and children, always been a total abstainer, no family history of insanity, fits, phthisis, or nervous disease. There is a large scar on the penis, admits having had syphilis and having been treated for it. He has enlarged glands in the groins. His illness commenced in 1895. He became irritable, falling into heedless passions, would get up at night and run out of doors, threatening his wife and children with violence. He then began to stagger in his walk, his speech became indistinct, and his memory weak.

History (obtained from wife, June, 1900).—H. E., now aged 42, occupation carman, married at 23, seven children resulted from marriage, five of whom are alive, and two dead, aged 11 months and 14 months respectively. Wife had one miscarriage. Six months before he went to Hanwell patient became very irritable and forgetful, nothing pleased him. He complained also of giving way of his legs, and of pains, which he thought were rheumatic, in his arms and legs. Pains were shooting in character. He complained also of pains in the stomach, with appearance of a lump that disappeared. Wife noticed that he had a difficulty with, and that there was a hesitancy in, his speech. Patient was a very temperate man. There is no history of insanity in the family on either side. Before his illness patient was a good husband and father, a strong man, of a very-easy-going placid disposition, and had nothing to worry about. He used to sleep well. She brought her boy to the hospital to see me, as she feared he might be going like his father ; the following notes were made :—

A. E., aged 13 years, the fourth child of the marriage. When he left school he was in the fourth standard. He was discharged from the Farm School for having stabbed two boys; he knew perfectly well what he was doing. Knee-jerks weak, only obtained by reinforcement, pupils equal, react to light and accommodation, no tremor in speech. The boy's mother notices no difference in him, he is good and affectionate. He suffers with headache now at times, affecting the back of the head, but he is never sick. His knowledge of time and place is good, simple judgment also good.

He laughs and talks in his sleep, and does not rest properly, but he never wets the bed. No history of fits. He has lately had a discharge from the ear; glands are a little enlarged.

September 26, 1900.—The following notes were made by me: About a year ago the patient had a congestive attack and remained in a dazed semi-conscious state for some days. He has had none since. He is childish, but has no marked delusions; his memory is poor, and his knowledge of time and place imperfect, but not absolutely lost. The speech is syllabic, hesitant, and slurred, tongue tremulous, and he has a mask-like expression denoting an emotionless state of the brain, but he occasionally bursts out into a childish laugh. He has an ataxic walk, the knee-jerks are absent, pupils medium sized, unequal, inactive to light, react to accommodation, complains of no girdle sensation, Romberg symptom well marked, considerable degree of hypotonus of muscles of the legs, the legs being capable of being bent at the hip to a right angle. He nowhere feels the prick of a pin, not even in the meatus urethræ, but responds with some delay to touch all over the body. When you prick him he says that it is a touch.

June 18, 1901.—Some time ago the patient commenced to have fits affecting the right side, followed by loss of use of the right limb, and the speech became even more indistinct and slurred. The hemiplegic condition has somewhat improved, but one can still see continuous involuntary twitchings in the right leg. Lying on his back he can voluntarily raise the leg from the bed, and he can use his arms without any marked incoordination of movement. The muscles are all very much wasted, and their power enfeebled, but the reason why he is unable to stand is mainly due to the ataxy. There is very marked hypotonus in both legs, and the deep reflexes are lost. He has no power over the bladder, and the water dribbles away continuously into the bed. Although the speech is so markedly affected, the dementia is rather childishness than anything else. He takes an interest in his surroundings, and an elementary knowledge of time and place is not wanting. He told me spontaneously that his neighbour had been in the asylum eighteen months, which was correct.

August 20.—A fortnight ago the patient had an attack of vomiting, very severe, which lasted for two days, accompanied with diarrhœa. He has not spoken since then. He is quite helpless, lying in bed on his back, did not recognise me, but he recognised Dr. Spark, and apparently made an attempt to speak. He is very emaciated, has complete loss of control over the sphincters, epigastric reflexes just present left side, absent right,

plantar reflexes present both sides. Pupils, right, 6 mm., left 5 mm.

The patient died September 10, 1901, of dysentery, of which he had suffered several attacks. The whole large intestine was in a state of acute and chronic ulceration. The brain, spinal cord, nerves, and muscles were removed by Dr. Bolton, and, owing to the softness of the central nervous system, the two former were placed directly into formaline, in order that they might be hardened before any descriptive examination was made. After a fortnight in this solution the brain and spinal cord were examined. The hemispheres show but little thickening of the pia-arachnoid generally, and there was not much wasting. Each hemisphere weighed 633 grammes; the cerebellum and pons, with the medulla, 140. There is some thickening of the pia-arachnoid over the mesial surface of both frontal lobes, especially the left. There is also thickening of the membranes over the left hemisphere in the following regions: the lower part of the Rolandic area, the adjacent Broca convolution, and the anterior portion of the first temporal. There is wasting of these structures, as is shown by the fact that the anterior portion of the Sylvian fissure is wider on the left than on the right side. It is covered, moreover, with thickened pia-arachnoid membrane. The bases of the first and second frontal convolutions and the adjacent portion of the ascending frontal are obviously wasted as compared with the right. The intervening sulci are deeper and the covering pia-arachnoid thicker and more opaque. The lateral ventricles are apparently not much dilated, but the ependyma is slightly granular, likewise the ependyma of the fourth ventricle. The spinal cord appears smaller than natural, but does not look flattened posteriorly. The posterior roots in the dorsal and lumbo-sacral regions, especially the latter, appear smaller and more gray and translucent than normal. On cutting the cord transversely, obvious naked-eye degeneration is visible in the posterior columns, exhibited by gray-red translucency as compared with the dead white of the antero-lateral columns.

Case 56.—*Tabo-paralysis of motor type, which commenced with occasional attacks of transitory loss of speech. History of insanity in family. Grandiose delusions and alteration in manner and disposition. Admission to asylum, on account of acute maniacal symptoms, development of motor paresis and ataxy; subsidence of mental symptoms, very little dementia.*

A. A., admitted to Hanwell, May 4, 1901, aged 40. Occupation, for a long time manager of Sanger's Circus. Has travelled

all over the world. Latterly, since he has lost his place, which has worried him a great deal, he has been a string bag maker. He has been married eleven years, no children, no miscarriages, steady and temperate. Father and paternal grandfather were insane.

History of present illness (from wife).—Two years ago he suffered with occasional attacks of transitory loss of speech and a bewildered look. Two or three months ago he became altered in manner and disposition, turned against his wife; and imagining he possessed thousands of pounds, indulged his extravagant ideas by buying useless articles.

Physical condition.—June 28, 1901. Walks with a wide rather ataxic gait, knee-jerks not obtained, some unsteadiness with the eyes shut, expression rather emotionless, tremor of tongue and face muscles, marked slurring and hesitancy in speech, pupils unequal, right 6 mm., left 5 mm., inactive to light, active to accommodation. Well marked syphilitic history, parchment scars on the body. There is a suppurating corn on the right little toe, now healed; marked hypotonus of hamstring muscles, no loss of joint sensation, no analgesia.

Mental condition.—He shows very little mental defect, he is able to give a rational account of himself and his doings in the past. He has no grandiose delusions now, and his knowledge of time and place are only slightly defective. There is no incoherence in his conversation, in fact, his disease appears to have affected especially the motor tracts.

July 4, 1901.—Pupils the same, mental and physical state unchanged. No cutaneous anæsthesia of thorax.

October 4.—Simple-minded, officious, declares that he feels quite happy. He has a perforating ulcer on the right foot, which causes him a lot of trouble at times.

October 30.—Perforating ulcer of right ball of little toe, also corn over ball of great toe. Speech much affected. No loss of joint sensation, no analgesia of feet and legs, no thoracic anæsthesia. Pupils equal, right slightly irregular, no reaction to light, slight reaction to accommodation. No attacks of sickness, no bladder trouble, Romberg symptom slight. Twenty years ago he had syphilis; there is a scar of an old gumma on the right thigh. Deep reflexes of legs lost. Mentally he is only slightly exalted; the facial expression also denotes exaltation of only a slight degree. He is clean in his habits.

June 19, 1902.—Suppurating corn on right little toe. He has no loss of joint sensation and no analgesia.

April 9, 1902.—*Physical condition.*—He walks with a slightly ataxic gait, the feet apart and bringing the heels down first. Knee-jerks absent both sides even with reinforcement. Lying in bed on his back, weakness of dorsal flexors of both feet noticeable in synergic actions of flexion of hip joints. Hypotonus of hamstrings equal on the two sides. Slight loss of sense of position in feet. None in hands. No loss of joint sensibility detected in feet or hands. No cutaneous anæsthesia or analgesia. No shooting pains in legs, no girdle sensation, no bladder trouble. All superficial reflexes present, also deep reflexes in arms. Sways a little with eyes shut, but can stand, though with some difficulty, on one foot. Speech is markedly affected, tongue and face muscles tremulous. Vaso-motor-paresis of vessels of face. He has lately had a number of congestive and epileptiform seizures.

Mental condition.—He is not the subject of delusions or hallucinations and he is only slightly demented as he can converse on most subjects rationally and his memory is good. He told me that when he reads it is not necessary for him to read the words silently ; he can do it by visual impression only.

Case 57.—Tabes following injury, modified gastric crises, causing delusions that his stomach had been swollen to enormous size. Death from tabo-paralysis.

L. H., aged 35. Admitted February 16, 1902, died May 31, 1902. Dock labourer, married.

Dr. Jones' note (February 21st, 1901, Claybury Asylum).—He is suffering from tabetic paralytic dementia. Mental reaction slow, and he has impaired memory, loss of knowledge as to time and place. He is somewhat depressed, and cannot realise where he is. He does not think there is anything the matter. He tells me he has six children, but he shows no concern to get to them and look after them. There is marked dementia. He is in fair nutrition, health impaired. Right pupil reacts slightly to light, the left less ; both are irregular, the right rather smaller. Knee-jerks absent. Common sensation in the legs retarded, cannot tell quickly when pin-pricked. Cannot stand blindfolded, and there is a tabetic gait.

January 9, 1902.—Mental reaction slow, is entirely lost to his surroundings, expresses delusions freely, says he is being worked on by electricity. He is fairly well nourished, has symptoms of tabes dorsalis. Knee-jerks absent, pupils react sluggishly, marked incoordination. Very fine facial tremors. Romberg's symptom.

History (from wife).—Dock labourer. Married twelve years. Four children alive, two dead, the first and last, eldest living 11 years, youngest 3½ years. Sister suffers with epilepsy. Seven years ago he had an accident, a bag of sugar weighing 2½ cwt. fell across the back of his neck. He was in London Hospital two days. Unable to work for three months, and received £15 compensation from his employers. Three years after the accident he commenced to have rheumatic pains, and pains in the stomach which were thought to be dyspepsia at the London Hospital. Complained of his head, and was very irritable. Always a man of moods ever since she knew him. He has experienced difficulty with his water and bowels, being very costive. He would go without food for days because of pains, but was not sick. He always said he felt as if something was drawing him in. Complained of crawling sensations. He had a kind of fit eighteen months ago when he was taken into sick asylum, then he tried to get out of the window. Transferred to Poplar, and then here. He was a good husband and a good father. Frequently visited by wife, who noticed that his mind became weaker each time. He had no fits that she knew of. Always complained of the pains and the dyspepsia, even recently when she visited him. Very much wasted. Syphilis doubtful. (F. W. M.)

Medical certificate.—He is under the delusion that his stomach has been swollen to an enormous size, also that people have conspired to put him away. At times he is quite lost in his mind.

Communicated by wife.—Strange in his manner, rambles in his statements. He was under the delusion that his body became swollen and of an enormous size.

Post-mortem notes.—State of nutrition and muscular system emaciated. Syphilis, no visible scar on penis; history of one in private notes made by Dr. Bolton. Skull somewhat dense. Average frontal thickness 6 mm., parietal thickness 3½ mm., occipital thickness 7 mm. Pupils equal. Subdural space.—Marked excess of fluid. Pia.—A little fronto-parietal milkiness, marked prefrontal adhesions and tearing of cortex on separating prefrontal lobes. Encephalon.—1,340 grammes, exceedingly œdematous. Weight of right hemisphere, 565 grammes; weight of left hemisphere, 550 grammes. General wasting of convolutions, obscured by œdema. Ventricles immensely dilated and granular. Cerebellum weight, 170 grammes, œdematous. Fourth ventricle. — Markedly granular, especially calamus. Cranial nerves.—Œdematous. Spinal cord as small as that of a child of

two years in the dorsal and lumbo-sacral regions. Obvious gray atrophy of posterior column, but no flattening of the posterior surface, soft meninges opaque and thickened. Thorax.—Bronchi a little congested. Bronchial glands œdematous. Right lung, weight 535 grammes, congested and broncho-pneumonic. Left lung, 415 grammes, more congested and broncho-pneumonic than right. Heart.—A little wasted, weight 205 grammes. Aorta and great vessels.—A small amount of early atheroma. Abdomen.—Liver weight, 1,150 grammes, much congested, density increased. Spleen, weight 78 grammes, density much increased. Right kidney, weight 95 grammes, cortex 4-5 mm., density increased; strips fairly readily. Left kidney, ditto. Abdominal aorta.—Slight early atheroma. Stomach.—Patchy congestion and chronic catarrh, also fibrosis. Small intestine.—Chronic catarrh and patchy congestion. Large intestine.—Considerable patchy congestion and catarrh. Abdomen.—Immensely distended.

Cause of death.—Broncho-pneumonia and bed-sores. General paralysis.

Microscopical examination of the brain (by Nissl method).—Ascending frontal and parietal, and parietal lobule.—Acute and chronic cellular changes, glia cell proliferation, increased vascularity, the perivascular lymphatic sheaths distended and filled with plasma cells, also a number of lymphocytes. The vessels in the same instances are quite empty and collapsed, but pigment granules of altered blood corpuscles can be seen both free, and in the proliferated cells of the sheath. Many of the medium and larger sized pyramids show changes resembling acute experimental anæmia produced by ligation of the cerebral arteries. The cytoplasm and nucleus are swollen, the latter is clearer than normal, eccentrically situated and surrounded by crumbling cytoplasm stained uniformly by fine dust like chromophilous particles. The processes of the cells are broken off and the cells are placed at all angles instead of in definite columns with their apical processes towards the surface. The small pyramids are similarly affected; only as a rule more cells have entirely disappeared. The intensity of the destruction varies in different situations in the same section. The cells of the molecular layer have entirely disappeared and there are numerous spider cells. There is cell proliferation, vascularity and thickening of the membranes. Some few of the Betz cells in the ascending frontal show marked chromolytic and nuclear changes. Some few are completely disintegrated, only a mass of very fine

granular cytoplasm remaining with crumbled edges. Besides the above portion of the cortex, the first and second frontal, Broca's convolution, central convolutions, posterior part of first temporal, angular and calcarine convolutions were examined. These all showed some of the changes described above, but the degree of severity of meningeal, vascular and cellular change is in the order mentioned, the changes in the occipital being very slight, for there is no meningeal thickening, and little or no abnormal vascularity, perivascular lymphatic dilatation or cell proliferation and but little dislocation of Meynert's columns. Some cells show chromolytic changes, but the acute changes are much less obvious than in the fronto-central convolutions. The changes noted above are in all respects like those met with in an ordinary fairly rapid case of general paresis.

GROUP 5.—Optic Atrophy and Tabo-Paralysis.

Case 58.—*Tabes, optic atrophy. Herpes zoster twelfth dorsal, tremor of tongue and lips, speech affection. Paralytic dementia in a man aged* 26, *seven years after infection.*—(*October* 7, 1900, *Charing Cross Hospital.)*

R. M., aged 26. Labourer, previously leather dresser. No other neuropathic history, except uncle died in St. Thomas's Hospital of nervous disease. Married five years Christmas, three children alive, one dead, lived one month. Twelve months ago patient saw double, and since then has suffered with shooting pains in the head, arms and legs, and pain around back. Eight months ago, suffered with weakness in bladder, cannot hold his water. Six months ago had difficulty in walking, especially in the dark. Eight years ago, chancre which lasted three or four weeks. Treated with iodoform at the Lock Hospital. No knowledge of any secondary symptoms. (It might be said that this was not syphilis, but I have seen so many cases treated for soft sore which afterwards developed brain syphilis, that I consider every venereal sore should be carefully watched and, if necessary treated. Only recently, within three months of infection, a case came to my out-patients', with optic neuritis and headache, and other signs of cerebral syphilis. He had only been treated locally. The patient recovered completely when placed upon mercurial inunction treatment.) Patient can stand with eyes shut and heels together, but cannot stand on toes. Cannot stand on left foot, but can on right. Knee-jerks absent. Pupils A.R., left 5 mm., right 4½ mm., and irregular in outline. Sexual desire lost four months ago, but was not excessive before

that time. Tongue and lips tremulous ; depressed anxious expression on face, and suffers with frontal headache. Articulation fairly good. Has shooting pains in the back of ear. Patient has not slept well, but has had no dreams.

Physical examination.—Old scar on dorsum of penis ; *glands shotty in neck and groins.* Strength of muscles good. Marked hypotonus, extended legs can be moved to a right angle with body. Plantar, cremasteric and epigastric reflexes present. No loss of sense of position of hand. Tactile sensation good. In left foot and lower part of leg some pricks are felt as touch, or give the sensation of contact with a cold body. Pins and needles or shooting pains in legs and arms. No muscular wasting. Slight loss of sense of position of lower limbs. Some slight thermo-anæsthesia in legs and both feet. Nearly blind in both eyes, but the left is more affected than the right. Primary optic atrophy both sides.

July 3, 1901.—Still pains in legs ; quite blind. Expression emotionless. Marked tremor in upper lip and tongue, slight hesitancy in speech, but no slurring. Has no fits, but suffers with headache occasionally. Still complains of weakness of bladder. Can stand with heels together, but cannot stand on one foot ; no ataxia in walking. Pupils right 5 mm., left $4\frac{3}{4}$ mm., both irregular in outline and inactive to light and pain, but react to accommodation and on convergence. On outer peroneal surface of right leg there is a patch of light tactile anæsthesia, a few spots scattered about on feet and legs where he does not appreciate a prick from a touch. Scars of herpes discovered right side area of twelfth dorsal. Superficial reflexes rather exaggerated. No thermo-anæsthesia. There is little change in sensory condition since he was here before. Although blind, he imagines that he sees people walking in at the door, and tries to get out of the way for fear of colliding with them. "He has got mixed in his speech for the past few weeks."

Case 59.—*Ataxy, optic atrophy, loss of memory, melancholia, visual hallucinations, delusions about his food, death from tabo-paralysis.*

W. B. H., age 45, single, draper's assistant, born in London, apprenticed at 16. At age of 30 contracted syphilis. Chancre (hard), followed by sore throat and falling out of the hair. He was treated for three months, but the gums were never sore. November 18, 1896, noticed dimness of vision of right eye, then

lost his sight in fourteen days. Previous to this he had insomnia for a long time. A few weeks later the vision of the left eye grew dim, and gradually became worse, until now he is almost completely blind. There is gray atrophy of both discs. His memory has not been good for several years. Four or five months ago he began to get weak in his legs; he is able to stand, but reels. There is no loss or delay of sensation to be detected in the limbs. The plantar and abdominal reflexes are brisk. The knee-jerks are absent. There is no local muscular wasting. He is emaciating, but he does not refuse his food. He can get up himself. The pupils do not react to light, are small and irregular; they react when he is told to look towards his nose. He has difficulty in micturition, but this may be explained by the fact that he has a stricture.

Mental condition.—He has visual hallucinations, and is very depressed. Painted up women, perfumed, come to him at night and taunt him. His facial expression indicates mental depression; the lines are partially obliterated. The tongue is a little tremulous, there is no facial tremor. His memory of past events is fairly good, of recent events not good. There is a little hesitation in speech, but no syllabic difficulty. He cannot remember simple sentences which he is asked to repeat, *e.g.*, one has to repeat word for word "The Irish artillery extinguished the conflagration," although he has not much difficulty in saying the words.

August 10, 1898.—He complains of shooting pains in the stomach and in the legs. He can localise sensation of touch and pain accurately. He knows when and how his great toe is bent. The knee-jerks are absent. The pupils are small, inactive to light, but react to accommodation. He has a delusion that he has never left Australia, and is still there, and he says that his sister has never been to see him, but that perfumed women come in to see him and make indecent overtures to him. His speech is slightly affected, being hesitant and tremulous. He says that his taste is good. Can hear a watch at one foot. No affection of taste, although he always says that there is something wrong with his food. He recognised salt and water perfectly.

Summary of notes of autopsy.—Dura mater adherent in front, pia-arachnoid is thickened generally, adherent slightly to the brain convolutions. There is some vascular effusion along the upper border of the hemisphere. Fourth ventricle is granular, there is general slight wasting of the cerebral gyri. The optic nerves are atrophied. Weight of brain 47½ ozs. Heart, left ventricle slightly dilated, first degree chronic mitral disease and atheroma of aorta.

Microscopical examination (by Dr. Hamilton Wright).—Central and Broca's convolutions; pia-arachnoid is thickened and its vessels congested. Nissl's method shows Meynert's columns in a disordered state, and a marked atrophy of many cortical cells. The processes of the latter are "corkscrew" and are devoid of chromophilous granules. Glia cells are exceedingly numerous and there are many spider cells present. The vessels are congested. The whole appearance of the cortex is as usually seen in cases of early general paralysis. Tangential fibres in Broca's convolution and the part corresponding to it on the right side are wasted.

Spinal cord.—Lower cervical region.—About one half of the fibres have disappeared from the posterior half of the postero-median columns. Many of these *in situ* are in a state of chronic wasting. There is marked atrophy of fibres along the anterior half of each intermediate septum. This atrophy passes backward, at the same time decreasing in amount, into the inner aspect of the base of each postero-external column. There is a slight degeneration of fibres in the root zones. All these parts are the seat of a sclerosis proportionate to the fibre atrophy. The rest of the posterior columns are practically intact. The roots external to the cord contain only a small number of unsound fibres. The posterior spinal ganglion cells of this region are healthy looking. The fibres from their distal ends are not obviously wasted. Thoracic region —The fibre atrophy and consequent sclerosis have practically the same distribution here as in the cervical region. From the middle of the postero-median columns it tends to spread laterally into the median parts of the postero-external columns. There is a slight degree of fibre atrophy in both root zones and in the roots external to the cord. The posterior spinal ganglion cells are not obviously wasted. The most marked feature is shrinking, probably artificial in origin. Lumbo-sacral region.—Wasting of proper fibres is marked along the postero-median septum in the upper lumbar levels; also in the middle portions of the postero-external columns. In the lower segments the wasting has the same distribution except that fibres in the position of the median oval area of Flechsig are almost intact. These pass backwards along the septum and tend to spread out along the periphery of the cord. The root zones are obviously wasted and sclerosed. Fibres in the position of the cornu-commissural zone are almost intact. There is a marked degeneration and denudation of root fibres external to the cord. Those still present are only in a few instances sound. Most are in some stage of chronic atrophy. Many cells of the posterior spinal ganglia in this region are in a state of fatty

degeneration, and so give a purple reaction to Weigert-Pal hæmatoxylin. The peripheral ends of the ganglia contain only a few wasted fibres in marked contrast to the proximal ends.

It will be seen by the above that there is a moderate degree of wasting in, and a consequent sclerosis of, those parts of the posterior columns which contain exogenous or root-fibres. The endogenously derived tracts, *i.e.*, the cornu commissural, descending comma, Flechsig's median, and Gombault and Philippe's tracts are practically healthy. The same may be said of the postero-internal zones. There is an intense congestion of all spinal, and spinal ganglion vessels. In the sclerosed areas these are slightly thickened.

Case 60.—*Optic atrophy, followed by tabes dorsalis, and later general paralysis.*

G. C., aged 36, porter, admitted May 27, 1894, to Marylebone Infirmary. Four years before admission began to suffer with giddiness, bad sight, and squint. One year ago began to suffer with a staggering gait and also dropping things, for which he lost his situation. Rheumatic fever several times. Denies syphilis. Jaundice twenty years ago, and at that time was crushed in a crowd, which caused him to be laid up for some time with a pain across his back. Gait staggering, but Romberg's symptom absent. He says he has difficulty in walking in the dark. Cutting pains in the legs, which are most severe in the left. Suffers with severe abdominal pains at times, probably gastric crises.

Eyes.—External squint of left eye. Movements outward incomplete. No reaction to light or accommodation. White atrophy of both discs. Knee-jerks present, ankle clonus. Tenderness over spine in the first dorsal, eighth dorsal, and lumbar region. Muscles good nutrition, no wasting. Sensation impaired all over face, chest, front, and both arms, with the exception of outer side of left arm ; sensations to cold also slightly impaired ; to heat, good. Leg sensation to touch almost absent, with the exception of the soles of the feet ; sensation to heat impaired. Both feet and hands are very cold. Bladder, at times cannot pass water. Colour vision very imperfect ; pink he calls yellow, violet black, cannot pick out green and calls it drab, chooses yellow correctly ; picks out mauve as being the nearest approach to red. Seen by Dr. Beevor on October 27. The case was diagnosed by him as one of posterior lateral sclerosis, with prominent hysterical element. "The ankle clonus and generally increased reflexes were those of an organic lesion. The changes could only

be of similar origin. The alterations of sensation were probably almost entirely hysterical." Discharged December, 1898. For the above notes I am indebted to Dr. Lunn. Patient eventually died two years later at Claybury Asylum of general paralysis and septic meningitis.

Case 61.—*Optic atrophy, preataxic stage of tabes several years, attack of mania, grandiose delusions, epileptiform seizures, death, characteristic brain lesion, arrested cord lesion of tabes dorsalis, degeneration of crossed pyramidal tracts, heterotopia.*

J. W., age 37, agent, sent to me by Mr. Gunn from the Westminster Ophthalmic Hospital as a case of tabes in the preataxic stage in 1893; attended my out-patient department, Charing Cross Hospital, for eighteen months. During that period there was little change in his condition. He presented the following symptoms: Failing sight, concentric limitation of the fields of vision, small pupils, unequal, A. R. No ataxy, Romberg's sign not obtained. Shooting pain in legs, absent knee-jerks. Cutaneous sensibility was not noted. No signs of mental affection. I lost sight of him, but eventually I remembered the name when I was taking his blood pressure some considerable time after his admission to Claybury Asylum in December, 1896, suffering from mania and general paralysis.

Family history (from brother, December 5, 1898).—Father intemperate; no consanguinity, no insanity. Patient has lived a very fast life with loose women, and has suffered with syphilis; latterly has had business worries, and six months ago he was very depressed and threatened to cut his throat.

Physical condition.—Knee-jerks absent, walks with a shuffling gait, tremor of tongue and lips, speech slurred and syllabic, exalted expression.

Mental condition.—Impaired memory, no idea of time or place, indifferent to his personal appearance and surroundings, delusions of wealth.

The dementia continued and progressed, and he had many epileptiform and congestive seizures, in one of which he died, May 14, 1899.

Autopsy.—Nine hours after death. Body emaciated, left pupil 2 mm., right 3 mm. Legs are flexed at thighs and knees, there are large bed-sores over sacrum and great trochanters; he has numerous small, symmetrical, papery scars on both shins. The calvarium is thin. bloodless, frontal bone 4 mm., occipital

6 mm. The pia-arachnoid was markedly thickened over frontal and parietal lobes, and slightly adherent to the cortex ; subdural space, moderate quantity of fluid. The basal vessels are the seat of a slight nodular arteritis. There is a slight wasting of first frontal and upper part of central convolutions on both sides. Cortical striæ are fairly well marked. The white matter and basal ganglia are slightly œdematous ; no sign of softening or induration. The optic nerves on both sides are markedly atrophied. Both lateral ventricles are slightly dilated, full of clear liquid, and moderately granular. The fourth ventricle is slightly dilated, but markedly granular. The spinal cord showed no naked eye change. Weight of right hemisphere, 563 grammes, left ditto.

Lungs.—Bilateral septic broncho-pneumonia, which was the immediate cause of death. There is nothing further of note in the other organs.

Microscopical notes (by Dr. Hamilton Wright).—Brain.—The posterior third of each third frontal gyrus was examined and found to be almost denuded of tangential fibres. Quite eight-tenths of them have disappeared. The cells are wasted and Meynert's columns disarranged. The pia is thickened and its vessels congested. There is a large increase in the number of glia cells. The whole cortex is like that seen in cases of general paralysis of moderate duration.

Spinal cord.—Cervical region.—The dorsal third of each postero-median column is slightly sclerosed. The sclerosis is most marked close to the median septum, and gradually diminishes in intensity as the intermediate septum is approached. The anterior third of these columns, except the apex, is also slightly sclerosed. A few fibres seem to have wholly atrophied in these patches of scar tissue. The great majority of those *in situ*, are irregularly swollen or attenuated. The postero-external columns along the outer side of the intermediate septa are slightly sclerosed, and a small number of fibres have disappeared. The most anterior parts of these patches of scar tissue widen out just before reaching the posterior margin of the healthy cornu commissural zone. There is no sign of atrophy in the fibres of the postero-internal zones. Charcot's root zones are intact in all segments of this region except the seventh and eighth. This is also the case in the extra-cordal portion of the posterior roots. In the seventh and eighth metameres there is a mild sclerosis and fibre atrophy in the root zones. A few fibres have disappeared from Lissauer's tracts. This, together with an atrophy of

fibres in the same positions in the upper few thoracic segments, is enough to account for the sclerosis along the external margins of the intermediate septa in the higher cervical segments. In both crossed pyramidal tracts there is a moderate fibre atrophy and replacement sclerosis; the intermediate gray matter through which the motor fibres course is intact. This appears to indicate that the degeneration in the crossed motor tracts is of fibres that end lower down in the cord. There is no atrophy in the anterior horns or roots. The central canal is normal. Thoracic region.—In the first two metameres the sclerosis of the posterior columns has practically the same distribution, and is of about the same intensity as in the seventh and eighth cervical segments. The sclerosis below the second thoracic plane is slight and diffuse in the postero-median columns, except their most ventral parts. There is also a mild diffuse sclerosis of the median aspects of the postero-external columns. The eleventh and twelfth thoracic segments are, in addition to the above, slightly sclerosed in the position of the root zones and Lissauer's tracts. No fibre atrophy or sclerosis is observable in the postero-internal zones, the comma tracts, or the cornu commissural zone. The lateral columns show a gradually increasing sclerosis as the cord is descended. As in the cervical region there is no rarefaction to speak of in the intermediate gray matter. Between the fourth and eighth thoracic segments the central canal of the cord is singular. It is irregularly cross-shaped, with short, transverse, and long, antero-posterior arms. The short arms pass off on each side between the gray commissures, and present a small bulbous dilatation at their extremities. The long posterior arm has pushed the posterior gray commissure backwards into the median septum for quite a fifth of the length of the latter. The ventral-ward extension has been turned to the left by the base of the anterior median fissure. Surrounding the enlarged canal is a thick layer of gelatinous substance. The cavity is narrow and free of the *débris* so commonly seen in the adult human cord. An even, compactly arranged layer of columnar cells lines the inner surface of the gelatinous substance. At first it was thought this state of the central canal was due to accidental distortion of the cord during removal. But the layer of columnar cells lining the cavity is compactly and evenly arranged, which could hardly have been so had there been an accidental heterotopia. Moreover, the canal is greatly dilated in the lumbar region, and has a projection backwards capped by the posterior gray commissure, as in the thoracic region. It appears to be a developmental defect. Lumbar

region.—There is a moderate, evenly distributed fibre atrophy and replacement sclerosis in the posterior columns of this region. It has scarcely affected the posterior aspects of the external columns, or the apices, composed of cornu commissural fibres. The fibres along the median septum are more closely arranged, and there is less sclerosis amongst them than in the rest of the posterior columns. The root zones and Lissauer's tracts are slightly atrophied and diffusely sclerosed. In the roots external to the cord are a moderate number of partially wasted fibres. A few fibres have apparently wholly degenerated. In the lateral columns one may see a compact sclerosis of the motor tracts. It is much more marked than in either the thoracic or cervical regions. In this region there is a noticeable rarefaction of the intermediate gray matter. A few irregularly swollen fibres pass from the lateral columns into it. No wasted cells are to be seen in the anterior cornua. Sacral region.—This does not differ materially from the lumbar region. The sclerosis of the posterior columns is perhaps proportionately greater and more general. Charcot's root zones and Lissauer's tracts contain fewer fibres than in the lumbar segments. The extra-medullary portion of the roots are as in the lumbar region. The sclerosis of the crossed motor tracts is quite obvious to the naked eye, and is, under the microscope, fully as marked as in the lumbar region. The pia-arachnoid is slightly thickened between the points of entry of the posterior roots in all the levels of the cord. But there is no special concentration of it where the roots penetrate the cord. Throughout the cord the blood-vessels are slightly dilated and choked with red discs. In the patches of scar tissue the adventitia of the vessels appears to be slightly thickened.

Case 62.—*Tabo-paralysis, commencing with optic atrophy, followed by slight ataxy, defective hearing, auditory hallucinations, deafness, history of fits, transitory aphasia, affection of taste and smell. Medullary, optic and spinal tabes, slight changes in cortex cerebri.*

S. E. J., aged 32, admitted to Hanwell, February, 1896, on the following certificate:—"He is rambling and incoherent in his statements; says he hears voices at night, and they tell him he must get up. He is at times very excited and answers voices which are talking to him." Occupation carman. The following history was obtained from his wife:—

Family history.—Father deaf and dumb, died of asthma (?), mother alive in good health, one brother in good health. No history of insanity, intemperance or consumption in the family.

Personal history.—He enjoyed good health up to six years ago, when he complained of pains in his legs. A little later his sight began to fail. This, however, did not prevent him from following his occupation for some little time. He attended the hospital in Queen Square under Dr. Buzzard, and the appended notes indicate his condition in April, 1893, showing that it was a case of optic tabes, in which the patient remained for some time in a pre-ataxic condition. He has been very temperate, and there is nothing in his occupation which might have led to his illness, with the exception that he has had boxes of oranges and potatoes fall on his head on separate occasions, once eleven years ago, and again seven or eight years ago. The brain affection appeared to have commenced twelve months ago with fits.

The following are the notes of his condition when under the care of Dr. Buzzard, kindly supplied to me by the registrar:—

Admitted into "Queen Square" on April 11, 1893. For two years and five months, dimness of vision of left eye. For two years and three months, dimness of vision with right eye, and vision with left eye almost lost. For two years, vision with right eye almost lost. Lightning pains in lower limbs (? date). One year and nine months, unsteadiness in walking. Nine months, shooting pains in back of head, both sides. One month, shooting pains across front of chest.

State on admission.—Gait slightly ataxic, also movements of arms. Shooting pains as above. Tingling in hands and feet. Sensibility to touch, pain, and temperature, good. Knee-jerks absent, also other tendon reflexes. Sphincters, unaffected. Vision, bad, only movement of objects. Right eye worse than left. Optic discs atrophic. Unequal pupils, loss of light reflex, nystagmus. Upward movement of both eyes and outward of left, defective. Hearing, right, $\frac{4}{4}$, left, $\frac{3}{4}$. Bone condition better on right side.

December 9, 1896.—He lies quite unconscious of everything around him. The attendant informs me that since I saw him, he has had many fits, which were more marked on the left side. Patient is greatly emaciated, and lies in bed with his legs drawn up, taking no heed of painful impressions, and apparently unconscious of his surroundings. When, however, food is placed near his lips with a feeder, he will suck at the spout, but does not

seem to distinguish between milk and a strong solution of quinine, which he takes equally well.

January 1.—Death from asthenia.

Present state (February, 1896).—He admits having suffered with syphilis before marriage. Physical condition.—His gait is not ataxic. He can stand with his heels together and his eyes closed. He is quite blind. The pupils are unequal, the right dilated, the left contracted, neither react to light. He suffers with pains in his arms, and he cannot touch the tip of the nose with his right forefinger, or make the two forefingers meet. He is deaf in both ears, but can hear loud shouting. There is nystagmus when told to look towards his right hand. There is no tremor of the tongue or face, but a little hesitancy and slurring of speech. He says he lost power of speech for some time last July. Examination of the fundus showed white atrophy of both discs, vessels normal in size.

Mental condition.—The memory is fairly good, but his intelligence is somewhat impaired. This may however be due to his blindness and deafness. He complains of hearing voices and sounds like bells ringing in his ears, especially at night.

July 8, 1896. —Progressive physical and mental enfeeblement. Painful sensation blunted, likewise taste and smell.

Abstract of notes of the autopsy from post-mortem book.—Pia-arachnoid opaque and thickened, especially over left angular gyrus. It is adherent to the brain substance in a few places. There is some congestion of the veins. Pia-arachnoid in the interpeduncular space is greatly thickened. Optic nerves are gray and shrunken. The lateral ventricles are dilated, but apparently not granular. The fourth ventricle is dilated and granular. There is a clot of blood outside the membranes in the lumbo-sacral region of the cord. The liver is fatty. Hypostatic pneumonia of the right lower lung.

Microscopical examination (by Dr. Hamilton Wright) : *Brain.*—The tangential fibres are almost wholly absent from the molecular layer of the central gyri. Most of the proper cells of the cortex are extremely atrophied. Glia and spider cells are greatly augmented. Spinal cord.—Vessels congested, great numbers of leucocytes, walls of arteries thickened. Cervical region.—The posterior median columns show a marked scattered atrophy of fibres, about one half have disappeared ; those in situ are fairly sound. On the outer side of each intermediate posterior septum is a band of sclerosis containing only a few fibres, all in a state of chronic wasting. This sclerotic band extends backwards

almost to the periphery of the postero-external columns. It is cut off from the commissure in its anterior aspect by fibres of the cornu commissural zone. Lissauer's tracts on both sides are almost denuded of fibres, and they have been replaced by scar tissue. The root zones of Charcot are deeply sclerosed. External to the cord the posterior roots are almost wholly atrophied. Many fibres remain in the bases of the postero-external columns, *i.e.*, in the position of the postero-internal zones. The fibres of the cornu commissural zones are almost intact. There is a readily observable atrophy of the plexus of fine fibres in the upper part of Clarke's column. There is no atrophy of fibres in the lateral columns. Thoracic regions.—The greatest atrophy of fibres is in the root zones, and middle third of the postero-external columns. There is, however, as in the cervical region, a considerable scattered degeneration and replacement fibrosis in both postero-median columns. Lissauer's tracts contain many healthy fibres, but a chronic atrophy of some of them is obvious. Outside the cord the roots contain only a few fibres. The cornu commissural zone and the descending comma tracts are pervaded by a slight sclerosis, and appear to have lost a few fibres. The great majority are present, however, and are healthy. The plexus of fine fibres around the cells of Clarke's columns are few in number. There is no special fibre atrophy in the antero-lateral columns. Lumbar region.—Charcot's root zones are greatly sclerosed, also the middle third of the postero-external columns. There is a marked scattered degeneration throughout the postero-median columns. Most of the fibres in *situ* lie close to the median septum. The latter feature is well marked in the lower lumbar regions. The fibres of the cornu commissural zone are present in large numbers, and are mostly sound. In the roots external to the cord there is considerable atrophy of fibres, but this is not nearly so marked as in the cervical and thoracic regions. The posterior spinal ganglia contain many cells in a state of fatty degeneration; others are excessively pigmented and devoid of chromophilous granules. The cell capsules are markedly thickened. There is a marked sclerosis of the substantia gelatinosa centralis about the central canal of the spinal cord, in the first cervical segment, and where it opens out to form the calamus scriptorius. The floor of the fourth ventricle is likewise sclerosed, up to about the level of the striæ acusticæ. The post-pyramidal nuclei show changes. A few cells appear to have wholly atrophied, and most of those in situ are shrunken and closely embraced by the replacement sclerosis. There is total atrophy of the plexus of small

fibres, and of most of the larger fibres, that usually surround or penetrate the nuclei. A marked atrophy is observable of the internal arciform fibres and fibræ rectæ, and there is a noticeable substitution sclerosis in the raphé. On both sides the nucleus arciformis is denuded of fine fibres, and its cells are shrunken. None of them, however, seem to have disappeared. In the corpus restiforme there is a slight diffuse sclerosis, with more marked islets here and there. It is possible that this is due to an atrophy of the continuations of the wasted arciform fibres. There is an almost entire absence of fine fibres from amongst the cells of the tenth, eleventh, and twelfth cranial nuclei. It is difficult to decide if there has been an actual destruction of any cells. Those present are shrunken and closely surrounded by an increased amount of sclerotic tissue. The degeneration in the intramedullary portion and in the stumps of the tenth pair of nerves is striking, but it is more marked on one side than the other. About one third of the more deeply implicated nerve has wholly atrophied. The complete atrophy of fibres is not so marked on the opposite side. Many of the fibres still present on both sides are irregularly swollen. In both the stumps and intramedullary portions of the nerves there is a considerable replacement sclerosis. As the hypoglossals pass ventral-wards to escape from the bulb, and in the stumps of their trunks, a few degenerated fibres may be observed. One of the most interesting features about this case is the atrophy in the nucleus ambiguus, or accessory vagal nucleus, and the solitary fasciculus on both sides. In the nucleus on both sides there is a marked atrophy of the fine plexus of fibres amongst the cells and of the fibres that compose the stalk. The more deeply implicated nucleus and stalk is on the same side as the more atrophied tenth nerve. Beyond doubt the marked atrophy in the tenth nerve is in part due to the atrophy of those fibres of the accessory vagal nuclei which enter into their formation. The vertical fibres of the solitary fasciculi are obviously wasted on one side only. On both sides the plexus of fine fibres in the surrounding gelatinous gray matter are wasted, almost wholly on the side where are the atrophied vertical fibres, and moderately on the side where the vertical fibres are intact. The more degenerated fasciculus is on the same side as is the least affected accessory vagal nucleus. Strands of fibres pass from both fasciculi to the intramedullary trunks of the tenth pair of nerves. They are not obviously wasted. In the ascending roots of the fifth pair of nerves there are a few wasted fibres and a moderate amount of sclerosis.

Case 63.—*Optic atrophy and blindness at* 28, *preataxic stage eighteen years, ataxy for a long time, preceded by mental symptoms, several fits, hallucinations, and delusions of persecution, progressive dementia.*

J. M., aged 48, metal polisher. Wife has known her husband since the age of 18. Patient's mother died suddenly of a paralytic fit. He was very unsteady before the age of 21, when he married. Since his marriage, patient has drank. A son was born twelve months after marriage, and his wife had a miscarriage two years afterwards, and no children since. The son was healthy, but died aged 22, of diphtheria. At the age of 28 (1880), patient began gradually to lose his sight, and he became completely blind in about twelve months. The doctors told him that he had "white atrophy." At this time he was mentally unstable. During the last two years (1898-1900) patient's legs have seemed to give way under him a little. He used at different times to pray, swear, &c., but never wandered from home. He finally squandered his money and "should not have had the handling of it for a long time before this." Patient had a fit in December, 1898, and was admitted to Bethnal House, from which institution he was discharged in September, 1899. He was at home for five weeks, and during this time whilst in a fit "put his head through a pane of glass." He had weakness of the right side, and was very shaky afterwards. Before he had been at home a fortnight "he began to be afraid, and wanted to go back to the asylum." He was admitted to Claybury on November 8, 1899, and was certified as follows: "Aspect vacant and smiling, quite incoherent, does not know where he is, can give no account of himself, wishes to die, answers imaginary voices, has been noisy, shouting 'murder' and 'police,' and saying voices accuse him of stealing." On admission, the speech, tongue, lips and facial muscles were tremulous, the pupils were fixed and irregular, the right being slightly the larger, he was quite blind, and slightly deaf, and the knee-jerks were absent. He was depressed and very emctional when spoken to, he had delusions of persecution, and said people were trying to poison him. He was very confused.

Further notes.—March 21, 1900.—Knee-jerks absent, sensation apparently normal, stands and walks very well for a blind man, pupils irregular and immobile, 3 mm., the right slightly the larger, slight wobbling nystagmus, white atrophy of discs, memory very good, knows the day and year on which he was

born, his age, period of blindness, &c., and says he has been here twenty weeks all but two days. Knows the present date, &c.

October 23, 1900.—Knee-jerks absent, hypotonus to within 20° of the vertical, right pupil very irregular, and left somewhat irregular. They vary in size around 3 mm., sometimes the right and sometimes the left being the larger. The speech is tremulous and jerky, and drawling and typical of general paralysis. The tongue is very tremulous, there is right facial paralysis, and the right hand is slightly paretic. He is exceedingly dull mentally, and anxious to go home.

November 16, 1900.—There is hypotonus to within 15° of the vertical in both legs. The right pupil shows most irregularity, and is slightly the larger. There is marked tremor of the tongue and face, and the speech as before is typical of general paralysis. There is some right facial paresis, but no weakness of the arms. He is very active on his legs considering his condition. He feels "all right" and happy. He remembers quite well recent and remote events, and the present day and date. He knows "as he has a memory." He is very impatient of control, and dislikes being examined as "he is blind, and it is therefore unnecessary."

January 4, 1901.—He is now an advanced general paralytic, both the pupils are irregular, the right 4½ mm. and the left 3 mm. He is now very feeble and cannot stand or walk. He sits all day in a chair rubbing his hands over his head, or one hand against the other (he was a metal polisher). To questions, he drawls, "I'm all right," or "I forgits," he is restless and irritable if interfered with, wet and dirty in his habits, and quite helpless.

February 10.—Death.

Abstract of port-mortem.—Summary.—Gross signs of general paralysis with sub-dural hæmorrhage, old-standing optic atrophy, syphilis probable but not positive, bladder hypertrophied, acute cystitis, broncho-pneumonia.

Morbid histology.—Microscopical examination of the brain by Nissl and other staining methods for showing fibres exhibited the ordinary characteristics of advanced progressive paralysis. The spinal cord and cauda equina were examined by the Weigert and Pal methods. (1) Cauda equina.—Marked congestion of veins otherwise no change in the vessels or their roots. Diffuse atrophy an denudation of fibres in nearly all the bundles, both anterior and posterior but especially of the latter. Some degree of substitution fibrosis but not equal to the atrophy. (2) Second sacral and surrounding roots.—Atrophy of attached posterior roots, diffuse degeneration of exogenous fibres in posterior columns and cornua,

escape of endogenous fibres. Slight sclerosis in crossed pyramidal tracts. (3) Fourth lumbar.—Ditto. Crossed pyramidal degeneration much more marked; anterior roots show no obvious atrophy; posterior roots, on the other hand, fairly marked atrophy, one-third to half the fibres gone; diffuse but pronounced degeneration with neuroglia substitution of exogenous systems, with escape of the endogenous; anterior horn cells unchanged except for some chromatolysis. (4) Tenth dorsal.—Slight degeneration of posterior roots, more marked on one side. Slight atrophy of fibre plexus of Clarke's columns, degeneration and atrophy of fibres in middle of postero-external column, and of fibres proceeding to form Goll's column, viz., middle and long fibres are degenerated in proportion to affection of the posterior roots of the lumbo-sacral region. There is some atrophy of Lissauer's tracts proportional to the atrophy of fibres in roots. Eighth dorsal.—Ditto. The pyramidal tracts do not appear to be so obviously sclerosed as in the lumbar region. Sixth dorsal.—Ditto. Seventh cervical.—No degeneration of posterior roots, diffuse degeneration and sclerosis of Goll's column, slight degeneration and light diffuse sclerosis in tract lying outside of Goll's column and corresponding to fibres from first dorsal and eighth cervical. No obvious degeneration of pyramidal tract commensurate with what is seen in the lumbar region. Second cervical.—Ditto. No obvious degeneration of direct tract at this level or elsewhere.

GROUP 6.—Conjugal Tabes and Paralysis.

Case 64.—*Conjugal tabic general paralysis, history of injury to head when a child, damage of prefrontal region of left hemisphere. Illness commenced with optic atrophy and blindness, followed by visual and auditory hallucinations, delusions, ataxy of arms, and progressive dementia. Death from acute attack of recurrent dysentery. A large depression due to loss of substance of anterior portion of second and third frontal convolutions left hemisphere. Typical appearances of general paralysis of brain. Naked eye sclerosis of posterior column. Atrophy of the optic nerves.*

M. M. C., widow, aged 38. Admitted to Claybury, June, 1901. Husband, a soldier, latterly worked as carrier, who had fits, and finally went out of his mind and died in Shoreditch Infirmary. She was married a long time ago while she was in service. She has had no children and no miscarriages. She says that she has had a lot of trouble. Between 6 and 7 years of age she had a fall from the balcony, and there is a depression the size of a

shilling on the forehead 1½ in. above the centre of the left orbit. Her memory is very bad. She has had a feeling of a tight belt round her waist, and still possesses it. She has been unable to unbutton her clothes for some time past. Her speech is hesitant and slurred, there is slight tremor of the facial muscles, also slight tremor of the tongue. She says that she has had several times attacks of sickness. Pupils 6 mm., regular, inactive to light and pain. White atrophy, with cupping of both discs, vessels normal in size. Expressionless face, except when talking, when she is frequently moved to tears or laughter; she is very restless and difficult to examine. Continuous restless movement of the hands; no fits, says she has wasted. Knee-jerks + + right side, absent left side, plantar reflexes + epigastric + +, no hypotonus in the legs. Skin sensibility.—Very difficult to test, apparently hypalgesia from third to the seventh segments inclusive. Some loss of power of localising, as she does not always put her finger accurately on the spot pricked or touched. Says that she has frontal headache, sometimes very bad. She has had a deal of worry. She does not care about food much, and is troublesome about it. Urine normal, temperature subnormal. She is very restless at night. The certificate stated that she was restless at night, and believed people came into her bed. She hears voices.

History of patient (from mother).—Her daughter was married twelve years ago; the husband had been taken to Shoreditch Infirmary because he was out of his mind, he was too ill to move to an asylum, and he died there. She believes that his mental complaint was brought on by worry, owing to his wife becoming blind, and his losing his place at Carter Paterson's. He had been a long time in the army prior to marriage, and had served in the Artillery. He had been to India, and was said to have had sunstroke there. For years previously to his death he had been irritable, but after his wife becoming blind, he had treated her kindly. He was a teetotaler for years, but latterly had occasionally broken out in drinking. He had latterly several fits. There is no history of insanity in the patient's family, with the exception of religious craze (revivalism) in the grandmother; she was not, however, put into an asylum.

Dr. Macmillan informs me this patient was the subject of visual and auditory hallucinations.

During her stay in asylum her mental condition was one of depression. She rarely spoke to anyone; she was very suspicious, and had delusions of persecution. She would do nothing

she was told, had to be dressed and had to be fed, as she believed her food was poisoned. She frequently complained of feeling "bad smells," and would often pull her clothes tightly round her and remark, "I'm in such a mess." She always complained of being dirty, and wanted to be washed. During the first three months of her stay here, she had the delusion that she was pregnant, but latterly seemed to have got rid of it. In January, 1902, she was transferred to the hospital for nine days, as she was suffering from an attack of dysentery. While there after the first two days she was much brighter, and developed the delusion that she possessed a large fortune. At the same time she was noticed to converse with "voices." She would sit up suddenly in bed, open her eyes and gaze at the ceiling, and, stretching her arms out, would say, "I am coming." She would often call on "Harry," and remark, "I will be with you soon." At times during her imaginary conversations, she would burst out laughing, as if amused by something she had heard. When allowed up, she would sometimes get up and walk across the ward, because she heard some one calling her to do so. If anyone spoke to her, she never turned her head towards them, but gazed up straight in front of her.

Abstract of post-mortem notes.—Patient died on April 15, 1902, eleven days after admission to Hospital from an attack of acute dysentery. The certified cause of death was, "Acute dysentery supervening on chronic."

Main facts of interest.—No external signs of syphilis on the body. Scar, the size of a threepenny piece on the cervix uteri close to junction with vagina which might have been the result of a primary sore. There was a hole the size of a sixpence in the region of the left frontal eminence closed by dense fibrous tissue to which the dura and pia is adherent. On removal of the brain the dense fibrous tissue had to be cut through and it was found there was a considerable loss of brain substance in the anterior part of the second and third frontal convolutions, the cavity extending nearly to the orbital surface of the lobe; it was large enough to have held half an ounce of fluid. There is almost generalised thickening and opacity over the pia-arachnoid of the anterior two-thirds of the hemispheres. There is some wasting of the convolutions in these regions. There is not much dilatation of the lateral ventricles. Ependyma granular, fourth ventricle very granular, especially about the calamus. After hardening in Müller fluid, naked eye degeneration of the posterior columns of the cord, with moderate atrophy of the posterior roots, especially

the lower cervical and upper dorsal, and lumbo-sacral. Marked gray atrophy of both optic nerves. Sections stained by Weigert method showed the usual degeneration of posterior roots and posterior columns of an early case of tabes.

Case 65.—*Conjugal paralysis.—Husband syphilitic, wife married twice, no children by second husband; the former died of tabo-paralysis, the latter a short time after of ordinary paralytic dementia.*

G. S., aged 43, docker, admitted to Cane Hill Asylum, July 19, 1899.

History.—Patient became gradually blind about two years ago, and subsequently suffered with locomotor ataxy, which took him to Stoke Newington Workhouse, where he remained one year. He then developed an attack of acute mania, becoming restless, excitable, and violent, threatening to stab another patient; he was certified insane. On admission to Cane Hill it was found that his wife was already in the asylum suffering from general paralysis. The following facts were elicited from friends: He did not drink, nor was there obtainable any hereditary history of insanity. He lost his sight gradually, and this was followed by drooping of the left eyelid, his mental state denoted considerable dementia and loss of memory, with defective knowledge of time and place; he has no delusions of grandeur or persecution, but is restless, noisy, and delirious, and although blind, has *frequent visual hallucinations.*

Physical condition.—He has a fatuous expression, the face is congested, the skin greasy and there is incomplete ptosis of the left eye (see photo., fig. 37). The pupils are unequal, dilated and irregular; there is primary optic atrophy of both discs and there is tremor in the lips and tongue, but not marked; the speech is somewhat hesitant and slurred; his gait is shambling; he brings his heels down first and he walks with a wide base, but there is no marked ataxy; the knee-jerk is *present on the right side, absent on the left.* He is too demented to give any reliable answers in regard to cutaneous sensibility; there are well-marked signs of syphilis on the body in the form of a scar on the glans penis, enlarged inguinal and cervical glands and tissue paper scarring all over trunk and limbs.

When asked about his wife, he says he does not know where she is and doesn't want to; she was brought to see him and she was much affected, but he was quite indifferent and told her to go away; he became more demented and died in December, 1899.

E.S., his wife, aged 39, had one child by her first husband, but no children by her present one; her illness began with a fit a short time before admission; she was taken to Guy's Hospital and thence to the infirmary, has had no fits since.

Mental condition.—She exhibits considerable dementia and is depressed, especially about her husband; the photograph shows this in her expression as compared with that of her husband.

Physical condition.—Pupils equal, react to light sluggishly; react well to accommodation. She can walk about, tries, though feebly, to make herself useful in the ward. Tremor of the tongue and lips, and slight paresis of the right side of the face. Speech is very tremulous and syllabic. Knee-jerks exaggerated both

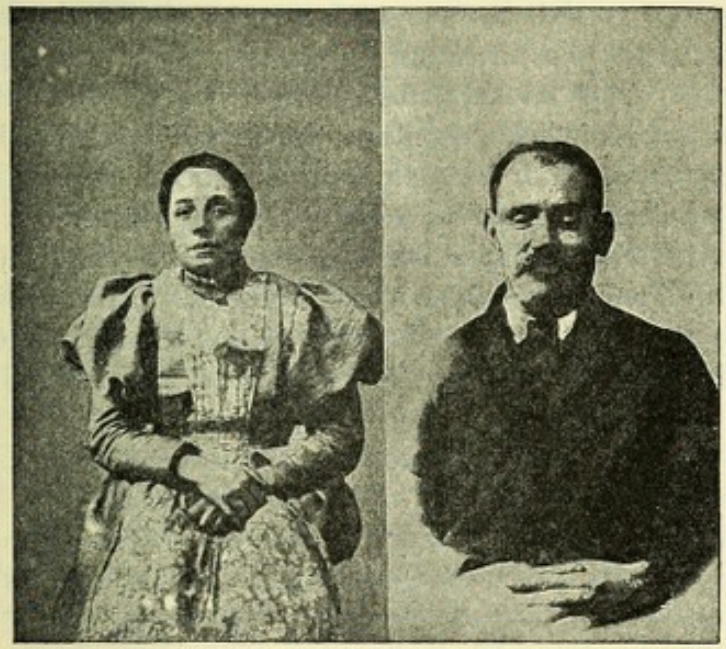

FIG. 37.

The physiognomy of the man denotes mild exaltation and dementia, that of the woman slight depression.

sides. The disease progressed, patient becoming gradually more demented; the tremor and speech affection more marked. The mental depression gave place to mild exaltation, and eighteen months after admission she died from exhaustion, following epileptiform seizures.

Post-mortem notes (abstracted).—An old scar found on the outside of the right thigh (? gumma), and cicatrix on the cervix uteri were the only signs which could possibly indicate syphilis. Body, well-nourished; brain, dura mater, non-adherent; considerable amount of fluid in sub-arachnoid space. Left hemisphere weighed 14 ozs., right hemisphere weighed 17½ ozs. The wasting of the

left hemisphere was especially marked in the prefrontal and central convolutions. The same applies to the right, but less marked. The pia-arachnoid is thickened, adherent, and opalescent. Both lateral and fourth ventricles are granular, and the left ventricle is much larger than the right, corresponding to the difference in weight of the hemispheres. There is early atheroma of the aorta, but the cerebral vessels are normal.

Post-mortem of G. S. (husband).—Died five months after admission. (Abstract.) Brain; pia-arachnoid thickened, especially over the frontal and central convolutions; atrophy of the left superior parietal lobule. Over the inferior surface of the orbital lobes on both sides, the brain substance, and the pia-arachnoid covering it, has an appearance not unlike that of a cirrhotic liver; on the mesial surface and tips of the prefrontal lobes, there is marked pia-arachnoid thickening and wasting of the convolutions. The lateral ventricles are not much dilated; the ependyma is somewhat granular; the fourth ventricle is dilated and very granular; Broca's convolution shows very little wasting; the pia-arachnoid strips with few erosions, the gray matter of the cortex is diminished, and in the lower part of the central convolutions the cortical striæ are ill-defined. Both optic nerves are greatly wasted. Left hemisphere weighs 19 ozs., right 19½ ozs. Pons medulla and cerebellum 7 ozs. Aorta was markedly atheromatous.

Death was caused by gangrenous pneumonia.

Microscopical examination of the brain (by Nissl, Marchi, Marchi-Pal and Weigert methods).—Sections were taken from (1) ascending parietal, (2) second and third frontal, and (3) orbital. *Summary of results.*—The changes are mostly of a chronic degenerative character in all the areas examined; few cells were acutely changed. The most marked acute changes in the cells are in the orbital region, where the vascular changes in the form of stasis and perivascular cell proliferation are most pronounced. The neuroglia cell proliferation is nowhere very marked. Recent degenerated radial fibres are seen in the central convolution by Marchi method, but none in the tangential or supraradial fibres. By Marchi-Pal and Weigert methods these layers of superficial association fibres are greatly diminished or absent in all those regions examined. The changes correspond to an ordinary slowly progressive case of dementia paralytica.

Examination of the spinal cord, ganglia and roots (by Marchi, Weigert-Pal, and Nissl methods).—By the Marchi method, sections of the cord in the cervical region at different levels showed a large

number of degenerated fibres scattered about in the posterior columns, also in the crossed pyramidal tract on the right side and the direct of the left, showing that there was a recent degenerative process occurring in the brain.

Different levels of the cord in the cervical, upper, middle, lower dorsal and lumbo-sacral regions were examined by the Weigert-Pal method. Sclerosis of the right crossed pyramidal tract and of the whole posterior median column was found in the cervical region. In the dorsal region there was degeneration in the postero-external column corresponding to Charcot's root zone. This was especially obvious below the eighth dorsal. It seemed in this region that the degeneration and atrophy of fibres was more obvious on the side opposite the degenerated pyramidal tract. The sections were cut to include the roots, and sections at one level would show far more degenerated fibres in the roots than at another level. The degenerated roots would be sometimes more apparent on one side than the other. Lissauer's tract generally showed diminution of fibres, but it varied at different levels. In the lumbo-sacral region, sections showed atrophy and denudation of fibres throughout the posterior column, except in the oval area of Flechsig and the cornu commissural zone. As in the dorsal region so here, the atrophy and sclerosis of roots is the subject of considerable variation, and is nowhere complete.

Spinal ganglia (stained by Weigert method) cut in series.—The cells show in great part some protoplasmic change, for they have a blue appearance owing to the presence of fine stained granules. The nerve-fibres leaving the ganglion show considerable atrophy and sclerosis. In the capsule of the gland, and in the distal roots, a number of bundles of fine medullated nerve-fibres are seen, which are either deficient in fibres or are unstainable. The third lumbar, stained by Nissl method (serial sections of this ganglion being made).—All the sections showed a variable number of cells presenting change from slight chromatolysis to a very marked condition of the same. In many of the cells the nuclei are eccentric and hardly stained at all.

Vascular and meningeal changes.—The changes in the meninges are not extensive, and although they are somewhat thicker over the posterior column of the cord, still there is not a marked flattening as is found usually in a prolonged chronic case. Nowhere could a process of endarteritis be seen, and the thickening of the vessels would in no way explain the atrophy of the fibres. The sections of the cauda equina show that the vessels in the undegenerated anterior roots were as often thickened as

those in the posterior, and in no case was an artery found blocked. Congested veins were found equally in both degenerated and undegenerated roots.

The retinæ and optic nerves were cut in celloidin and stained by logwood and eosin, by Ströbe and Weigert methods.

The retinæ exhibited no inflammatory changes or alteration of the vessels. The ganglion cell and nerve-fibre layers were entirely absent. The rods and cones were not distinguishable, but there was undifferentiated protoplasm, stained with the logwood, lying between the hexagonal pigment cells and the granular layers, which are intact. The sustentacular fibres of Müller were very distinctly seen owing to the absence of the ganglionic layer. There was considerable excavation of the papilla.

The optic nerves were shrunken to half the normal size. The nerve-fibres were absent, their place being occupied by a fine meshwork containing a large number of round and oval granular nuclei or cells. The interstitial septa are thicker and more obvious than usual, and contain also a number of proliferating cells or nuclei. The vessels show no apparent change or only slight thickening of their walls.

Case 66.—*Conjugal paralysis. Husband suffered with fits, hemiparesis, and dementia, sluggish pupils; diagnosed by Dr. Bailey and Dr. Alexander as probable general paralysis. Wife admitted with acute mania and died of tabo-paralysis.*

L. H., aged 54, admitted to Hanwell, January 11, 1901, suffering with acute restless mania.

History (from daughter).—Worried about her husband's illness, and for the last two years has been strange, easily excitable and always talking about her troubles. The husband had a fit two years ago, since then unable to do any work, and this preyed on her mind. Two months before admission she had a fit followed by left-sided hemiplegia of a transitory character, she remained in bed three weeks, after this she was able to walk about as before. She then became restless and strange, getting up in the night to hide things, wanting to buy all manner of things, going out of doors and talking to strangers about the way people treated her, untidy in her dress and dirty in habits.

When I saw her four days after admission she was in a state of acute mania resembling alcoholic mania, but there was no history of intemperance. There was a history of three successive miscarriages, and the notes state of syphilis. This was probable

from statements made in her delirium. She kept repeating, "he poxed me—my son must be cut like a Jew." She says she dreams all manner of things, she sees Old Nick. She says that they have put poison in her feet, legs, and arms. She says burglars came into the house, they boiled the pot and then poured it down her throat. She thinks she is at Crawford Street still. She is going to be married and wants clothes of gold. She does not recognise the nurse or doctor. She has not been to the closet for three months; the food is poisoned. Continuous restless motile delirium, and tears up her clothes. Sings and shouts and hardly sleeps at all while in the padded room. She was brought to the asylum tied on a stretcher.

Physical condition.—Emaciated, no teeth, knee-jerks absent, tenderness of the calves when compressed, pupils unequal R. > L. 4½ mm., 4 mm., speech a little slurred and hesitant.

March 17.—Mania still, but not so marked, yet restless, excitable and full of delusions about valuable ornaments and gold watches, frequently takes her clothes off, and is very troublesome. Arteries atheromatous; aortic regurgitation, collapsing pulse, acute mania of general paralysis diagnosed.

March 28.—Mania subsiding, orientation in time and place very imperfect. No tremor of tongue, articulation fair. Pupils irregular, dilated, equal inactive to light, sluggish to accommodation. No tenderness on pressure of legs. Knee-jerks absent. She now knows her friends and talks to them, but memory very poor.

October 30.—She is now a well pronounced general paralytic. Dr. Bailey saw Mr. H., the husband,[1] he is partially demented, pupils unequal, sluggish reaction to light, speech slow and hesitant; history of several paralytic seizures. Dr. B. considered that the patient was a general paralytic.

November 28.—Much weaker, confined to bed; sometimes hardly speaks for weeks together. To-day she was more talkative. Knee-jerks absent. Triceps-jerks easily obtained. Lies in bed with hips and knees semi-flexed, but feet plantar-flexed and somewhat inverted. She does not take any notice of sharp pricking of the legs, nor of a strong faradic current, but she immediately does when the arms are stimulated or pricked. All the muscles respond to faradism. Plantar and epigastric reflexes obtained with great difficulty.

December 6.—Right-sided epileptiform convulsions.

[1] Subsequently I ascertained from a doctor who treated the husband that he had had syphilis.

December 13.—Death. Pupils unequal, right 5 mm., left 6 mm.

Post-mortem.—Body much emaciated. Left hemisphere subdural hæmorrhage about one week old. No naked eye softenings, Right hemisphere 18 ozs., left 18 ozs., cerebellum and pons 5 oz. Sub-arachnoid fluid increased. Ventricles somewhat dilated, granular ependyma, especially lateral sacs of the fourth ventricle. Pia-arachnoid thickening over frontal and central convolutions especially. Some atrophy of convolutions. Typical appearances of early general paralysis. Aorta atheromatous second degree, and aortic valvular incompetence. Spinal cord—after hardening in Müller—showed well-marked posterior and lateral column degeneration.

Case 67.—*General paralysis* (*tabetic*), *wife suffering from locomotor ataxy. Impaired cutaneous sensibility to pain on the left side after a series of epileptiform seizures. Death one year after admission.*

W. F., aged 38. Occupation journalist, admitted to Cane Hill, December 6, 1900, first regarded as an epileptic. He came from Lambeth Infirmary under the following certificate:—"Strange in manner and states that he is starved here (a delusion); states he wants to go to South Africa at once before Roberts and De Wet finish; that he has the editorship of a leading paper there; says that he has a comfortable home there to go to, and plenty of money. The wife states that there is no home now." There is no history of insanity, phthisis, or alcohol. He has been married nine years, no children, one child born dead eight or nine months after marriage, denies syphilis, but there is a scar on penis and indurated glands. His illness commenced two years ago with a fit. At the end of January the patient had a series of left-sided epileptiform seizures which according to the attendant began in the angle of the mouth and spread over the left side. He does not lose consciousness but becomes drowsy. I saw him in June, 1901, for the first time. The attendant informs me that the wife, who lives at Sheffield, had visited the patient and he had noticed that she walked with a characteristic ataxic gait, and she told him herself that she had suffered for some few years past from the same disease, locomotor ataxy. Patient is lying in a semi-stuporose state being affected with a congestive seizure, so that he is incapable of examination.

July 5.—Patient is now up and walking about. The following notes were made:—

Physical condition.—There is no ataxia in his gait and he can stand with his eyes shut ; grasp good both sides, and there is no muscular weakness. No loss of sense of position in the hands or feet, superficial reflexes rather exaggerated, knee-jerks absent both sides, triceps-jerk and wrist-tap contraction easily obtained, no affection of speech or handwriting, tongue slightly tremulous, somewhat expressionless face except when conversing. Pupils equal, 3½mm., a little irregular outline, do not react to light or pain, react to accommodation. The last time I saw him the pupils were unequal, the left being the larger.

No trouble with the bowels or bladder.

The attendant says that he has had several left-sided seizures since I saw him, which last about one and a half minutes. In one, the attendant saw him go to the left in falling "like a peg top." Some hypotonus in the legs, rather more marked on the right than the left side.

Sensory.—Cutaneous Sensibility.—All over the left arm and leg and left side of trunk are spots in which prick of pin is not felt, or only as a touch ; there are also spots of tactile anæsthesia. He was tested in the following manner, stripped and eyes blindfolded. He was told to touch with his finger the part pricked or touched. This he invariably accomplished accurately and without delay on the right side, not so, however, on the left. Sometimes he localised wrongly, sometimes he did not feel at all the prick of a needle or touch with finger. There was no precise root area determinable except over the left buttock and around the anus on the side that the defective sensibility was most marked. No loss of joint sensation could be determined. He has no hemiparesis. Smell and taste.—Thinks asafœtida smells like violets, cannot smell rose water or strong peppermint. He did not recognise acids, salines, or syrup, but recognised bitters when quinine was painted on the back of the tongue. There is no limitation of the field of vision, and his colour perception is good enough to pick out the ordinary colours. Hearing.—Hears a watch at 18 ins. from both ears.

Mental State (October 15).—Patient is fairly coherent, except when suffering with conjestive seizures. He has had hallucinations of vision, seeing people lying alongside of him and annoying him. He is said to have had delusions with regard to his bowels. He can give a fairly rational account of himself, and I could find no gross loss of knowledge of time or place. He says that he has had no pains in the legs and no feeling like a cord round the waist.

January 13.—Death took place.

The brain showed well marked signs of general paralysis in the

first stage. First stage of sclerosis in posterior column. Wife was seen by Dr. Cribb, and he found her to be a well-marked case of locomotor ataxy. She had attended Queen Square for some years for this complaint. I wrote asking her to come and see me, but she did not turn up.

Case 68.—*Conjugal tabo-paralysis of an aged couple (the melancholic type). Wife affected after the husband, rapid course of the disease in both cases.*

E. E. G., admitted to Claybury, December 14, 1900, died October 19, 1901, aged 64 years; occupation washerwoman, moderate drinker.

Family history incomplete, husband a carpenter in Woolwich Arsenal, afterwards pensioned, was several years in the Volunteers, died in Barming Heath Asylum, July 26, 1899, after two years and four months residence, aged 64 years. Had two fits before he went away, and one later. He went blind while he was in the asylum, he was said to have "paralysis of the brain," or "general paralysis." Patient was married forty-five years, she had in all eighteen certain conceptions, two boys and five girls are alive and healthy. She had at least five still-borns or miscarriages, all the rest died as babies.

History of attack.—Patient grieved very much when her husband was taken to Barming Heath, but she kept fairly well mentally for some time. Twelve months before admission she became suspicious and strange in her manner; "one time said she was not at home and got her things, and said she was going home," very restless at night, began to "talk funny and say senseless things," used to hear voices about. If she had money, "they never knew where it went"; she thought "robbers were getting into the house," and "there was a cellar below, and children were crying in it." She could not be left alone; "one night she got out and had a clock under her arm and a cup of tea in her hand, and said she was going home." Her husband had been a very similar case, and as he had done, so used she to cry for hours at a time, and say that everyone was hard on her, and no one did anything for her, and also that she never had any money, and was not at home. Some months later she fell down in the back garden, and could not speak for twelve hours, but seemed to have no paralysis. She was then in bed for about six weeks. After this she was up for about a month, during which time she

began to wander about as before, or worse, and finally was sent away.

Certificates.—Very talkative, restless, and incoherent, is constantly getting in and out of bed at night, wandering about, talking to imaginary persons, and quarrelling with other patients and pulling them about.

On admission.—Serpiginous ulcer on outside of right knee (diagnosis syphilitic), scar on left leg, bronchial râles in chest. Memory much impaired, depressed and miserable, gives a confused account of her past life, sees strange persons about her bed, wet and dirty in her habits.

January 3, 1901.—Knee-jerks absent, right pupil 2, left $1\frac{2}{3}$, they accommodate to $1\frac{2}{3}$ and $1\frac{1}{3}$, right (?) irregular, left irregular, no reaction to light, very little tremor of tongue, speech not characteristic, talks rather sensibly but is demented, is occasionally wet.

March 10, 1901.—Brighter but memory remains defective.

September 10, 1901.—Palate high, teeth very good for age, considerable tremor of tongue, voice very tremulous and hesitant, and characteristic of general paralysis, right pupil $2\frac{1}{3}$, left 2 minus, accommodate to 2 and $1\frac{1}{3}$, both knee-jerks absent, left foot has a sore on the outer malleolus, and is redder and colder than right. The ulcer on the outer part of the right knee has healed leaving a serpiginous pigmented scar. Patient is in bed and very feeble. She is very emotional and cries when her husband's name is mentioned. She remembers my face though she has not seen me for eight months.—(F. W. M.)

October 19, 1901.—Died of exhaustion.

Post-mortem.—External appearances as described above. Skull.—Considerable hypertrophy of inner table in frontal region, and dural adhesions. Right pupil 4 mm., left 3, no definite signs of optic atrophy, but the left is a little smaller and grayer than the right. Great excess of subdural fluid. Pia-arachnoid, there is considerable fronto-parietal thickening and milkiness, much congestion, sub-pial extravasations on right pre-frontal region and scattered over left frontal region. Mid-line pre-frontal adhesions, which decorticate on stripping, slight excess of sub-arachnoid fluid, basal vessels much dilated, and internal carotids highly atheromatous. Weight of encephalon 1380 grammes, right hemisphere 575, left 580, cerebellum and pons 170. Moderate pre-frontal wasting, lateral ventricles much dilated and granular, third ventricle granular, fourth ventricle granular throughout. The whole of the encephalon is congested and the sinuses are full of blood-clot.

Lungs emphysematous and œdematous, heart flabby and full of dark clotted blood, cavities dilated, mitral valves slightly atheromatous, one or two specks of early atheroma in coronary arteries, a relatively little amount of second stage atheroma in the aorta and a few pearly white patches, which are more frequent in the abdominal aorta than in the thoracic. Liver congested but otherwise natural except for a little scattered fatty degeneration. Spleen small and diffluent; kidneys, capsule slightly adherent, congested, cortex 4-6 mm., density increased. Renal arteries considerably thickened, stomach catarrhal, intestines natural, subacute cystitis, uterus somewhat atrophic, ovaries and tubes extremely atrophied.

Cause of death.—Exhaustion of senile general paralysis.

Microscopical examination of spinal cord.—Size fairly normal at all levels, no flattening posteriorly, only slight pia-arachnoid thickening. Slight diffuse atrophy in direct tracts both sides, most noticeable in the upper cervical region. The crossed pyramidal degeneration is apparent to the naked eye in the lumbo-sacral region, but apparently diminishes as the cord is examined upwards. The exogenous fibres in the lumbo-sacral region are all markedly affected, and there is atrophy of the plexus around Clarke's column cells. The posterior roots are also sclerosed, and partially denuded of fibres in this region. In the dorso-lumbar region there is considerable atrophy, but the short, fine fibres are less affected. In the mid-dorsal there is very little root-fibre degeneration. In the upper dorsal there is slight degeneration of posterior roots, and cornu radicular one.

Copy of entries in Barming Heath case-book respecting G. H. G: Date of admission.—April 12, 1897. Age and sex.—61 years, male. Friend.—E. E. G. (wife), 39, H. Road.

Medical certificate (W. E. Boulter, April 10, 1897).—His speech is uncertain and hesitating, and he is generally tremulous. He says he has only been out of sorts a short time, is quite cheerful, and says he can still do his work all right, but people won't take it when he has done it. (2) His wife, E. G., 39, H. Road, says he has been very strange for the past eighteen months, has delusions that he is going to die at once, lays himself out in bed and calls all his children to him. He is much upset by work being refused that he thinks he has done properly.

On admission.—Mind a blank. Has no idea of time or place. Has no knowledge of where he is now, or where he has come from. Is happy and light-hearted, laughs stupidly like a drunken man, and says he feels as happy as a dog. Physically well developed,

well nourished. Lungs healthy. Heart irregular, aortic regurgitant murmur. *Is very ataxic.* Pupils irregular and do not respond to light. Speech thick and mumbling, will commence a sentence all right, then slur over half a dozen words, and finish off correctly. Knee-jerks absent, free from bruises.

April 27.—No improvement, marked signs of general paralysis.

May 14.—Advanced in general paralysis, much weaker.

June 10.—Very weak and helpless, dirty.

August 20.—Patient had a severe convulsive attack to-day.

November 20.—Patient has picked up again, but very tottery.

February 7, 1898.—He is getting much weaker, speech very bad.

March 8.—Very weak, advanced in general paralysis

April 8.—Very weak and uncertain on his legs. Pupils widely dilated. General paralysis well marked and advancing.

September 5.—Rapidly worse, hardly able to move. Very restless at night, generally sleeps in padded room.

December 8.—Unable to move or help himself, destructive.

June 27, 1899.—Very feeble and utterly helpless, no mind.

July 26.—Sank and died this morning at 6.15 a.m. in the presence of Attendant J. Brooker, the probable cause of death being general paralysis of the insane. One bedsore on right shoulder, one in the middle of the back, and a slight one over sacrum. Notice of death sent to Coroner this day, of which the following is a copy: G. H. G., male, 63 years, married, wood sawyer, admitted from 39, H. Road, died at 6.15 a.m., July 26, 1899, of general paralysis of insane (not ascertained by *post-mortem* examination) in the presence of Attendant James Brooker, the duration of the disease being about three and a half years. Bed sore on shoulder and middle of back, no mechanical restraint. (Signed) F. Pritchard Davies, M.D., Medical Officer; Thomas W. Dadd, Clerk (Dated July 26, 1899).

A *post-mortem* examination was refused (F. Pritchard Davies, M.D., Superintendent).

Case 69.—*Conjugal general paralysis in husband and wife. Definite signs of syphilis in the wife.*

E. C. R., aged 53, married; occupation, furniture dealer. Admitted to Claybury, September, 1896, with delusions and auditory hallucinations; he threatened to murder his wife; there is no history of insanity or intemperance, he was married twenty years

ago, and has one child. Exciting cause attributed to failure in business and family troubles.

Condition on admission.—There is well-marked leucoplacia of tongue, indurated glands in the groin, but no evidence of a scar on the penis. He is in poor bodily condition, the knee-jerks are obtained with difficulty, but no ataxy noted, the pupils are said to be equal, and to react to light and accommodation.

Mental condition.—He is talkative and incoherent, he complains of his wife's actions, and states that he wished to kill her and the boy. At times he has religious exaltation, and wants to die. He talks of his mother possessing a large property, and he is continually making large profits out of the exchange and sale of furniture. His knowledge of time and place is most imperfect. Occasionally he is noisy, excitable and difficult to restrain. Diagnosis, mania of general paralysis.

His general health improved in the asylum, and the symptoms of acute mania gradually subsided. About a year later a growth was found in the left half of the tongue which was called epithelioma; one half of the tongue and the sub-maxillary glands were removed. There was no recurrence of the growth. As no microscopical examination of this was made, it is open to doubt whether this was epithelioma; in fact, the history of leucoplacia, the syphilitic eruption and the syphilitic eruption on the wife, to be afterwards described, make it probable that it was a syphilitic gumma. The symptoms of general paralysis became more marked, tremor of the facial muscles, slurred speech, pupils inactive to light, and occasional epileptiform seizures with mental exaltation, firmly established the diagnosis. On October 12, 1898, he had an apoplectiform seizure followed by paresis of the left arm and leg, accompanied by pyrexia, 103° F. This is of interest in connection with the atrophy of the right hemisphere found *post mortem*. On November 13, 1899, he again had a seizure affecting the left side. It was noted that his speech was never much affected; in fact, the attendant says that his speech has never been any worse than one would expect in a man who had lost half his tongue. The *right knee-jerk just present, the left markedly exaggerated*, but no ankle clonus. Right and left triceps jerks exaggerated, all superficial reflexes increased. His mental and bodily condition progressively deteriorated, and he died on December 18, 1899.

Post-mortem examination showed the following conditions:—Enlargement of cervical and inguinal glands, but no other signs of syphilis, no evidence of recurrence of growth in mouth. Brain.—Adhesions of dura mater to skull, pia-arachnoid thickening, which

gives rise to erosions on stripping, marked wasting of the convolutions in the frontal region, and also of those along the vertex of the hemispheres adjacent to the superior longitudinal sinus. The right hemisphere weighs 90 grammes less than the left, the cortex is diminished, especially of the right hemisphere, and the striæ indistinct. The ventricles, both lateral and fourth, dilated and very granular. Weight of right hemisphere, 475 grammes, weight of left hemisphere, 565 grammes. Atheroma of aorta and coronary arteries, slight atheroma of cerebral arteries about base.

E. R., aged 37, housewife, admitted to Cane Hill Asylum, October 13, 1897, died September 6, 1898. Mental disorder, dementia, with general paralysis, certified cause of death general paralysis of the insane.

On admission the notes state that this patient was a fairly well nourished woman. She was suffering with a syphilitic, scaly eruption on the face and legs, and ptosis of the left eyelid, and had a vacant, emotionless expression. The pupils were unequal, right larger than the left, both inactive to light. Superficial and deep reflexes were increased, and the speech was hesitant, slurred, and indistinct, with tremor of the tongue and face muscles. Mentally she was dull and slow in thinking and answering questions. Her memory and intelligence were much impaired; she had delusions, like her husband, about furniture, for she believed the ward furniture was all hers, and that she could exchange or sell it. The dementia progressed, and on August 25, 1898, she had an attack of left-sided epileptiform convulsions, which lasted for a few days; the condition became worse, and she died from exhaustion on September 6.

Post mortem.—At the autopsy the dura mater was found adherent to the calvarium. The pia-arachnoid membrane thickened and opaque, adherent to the convolutions, which were atrophied and left erosions on stripping. The notes state there is considerable softening in the left hemisphere, chiefly in the frontal and parietal lobes, and an appearance of red softening. (No note is made of the condition of the veins opening into the longitudinal sinus, but it is probable that venous obstruction or thrombosis was the cause, and there is no doubt that the left-sided epileptiform convulsions, from which she had suffered just prior to death, were associated with and due to this condition). The right hemisphere weighed 18½ ozs., the left 21 ozs. No doubt an acute destructive process accounted for the marked loss of weight of the right hemisphere. The condition of the ependyma of the ventricles is not noted, but the lateral ventricles are said to be somewhat dilated.

GROUP 7.—Ataxy with Non-Progressive (? Alcoholic) Dementia.

Case 70.—*Ataxy, alcoholism, syphilis, neuropathic family history, delusions of persecution and hallucinations, probably the result of alcoholism and heredity, dementia, which combined with the physical signs led to diagnosis of tabetic general paralysis. Mental symptoms greatly improved, also physical.*

T. N., aged 39, admitted to Claybury, August, 1896, formerly a soldier, then commercial traveller, then kept a coffee house. Married, four children, two died a fortnight before birth, other two at birth, full-time. Lived a very dissolute life, given to drunkenness and great sexual excesses, acknowledges syphilis contracted at Aldershot, 1883, well marked scar, enlarged glands, secondary symptoms (treated twelve months), followed two or three years later by loss of sight in right eye. His wife states that he had marked satyriasis and had been unable to attend to his business, thrown about his money, slept badly, rambling round the house at night, and for days had taken nothing but beer.

Physical condition.—Tongue, clean tremulous; palate high; right eye blind, left good sight, pupils Argyll-Robertson; hearing good; deep reflexes lost; gait ataxic; fine tremor of hands; marked anæsthesia of legs, mostly on right side. Impaired sensibility of hands and face, "padded foot," girdle pains, has had gastric crises, and alcoholic gastritis.

Mental condition.—Great loss of memory, especially for recent events. Restless, and of dirty habits, and full of delusions. A little later, March, 1897, he is much distressed in his mind by fixed delusions of persecution. "His execution has been ordered, every hour is his last, so that he lives in continual dread that they are going to fetch him away to hang him."

July, 1898.—He is in good bodily health, but there is a recurrence of persecutory delusions in another form. "Lions and tigers are after him to devour him, and he begs for protection against them," causing him to be noisy and excited.

February, 1899.—Patient attacked by dysentery, from which he recovered. A later note states that he had swallowed stones with suicidal intent.

Health deteriorated, shows signs of tabes of a mixed type, anæsthesia and analgesia of both extremities and trunk, with thermal loss and complete loss of muscular sense—slight ptosis and paralysis of external rectus of both eyes—Romberg sign present, Argyll-Robertson pupils, plantar reflexes almost absent, abdominal brisk, knee-jerks absent.

Mental condition varies from time to time, sometimes much more lost, deluded, and incoherent than at others.

November, 1900.—His physical and mental condition had improved considerably. The memory is still bad, especially for recent events, but he has now no hallucinations or delusions and can talk in a fairly coherent manner and play a good game of draughts. He has also some knowledge of chess. There is much less ataxy and he does not sway when the eyes are shut. The anæsthesia and analgesia are much less and limited to patches on the lower extremities.

August 6, 1900.—Patient is quiet, has absence of all the deep reflexes, some hypotonus, no marked ataxy, no tremor in hands, can undress and dress himself, standing on one foot to put the other through the trouser leg, and able to put on his trousers himself, standing, so that there is very little unsteadiness of position. He can feel light tactile impressions correctly all over the body with the exception of a patch about the size of a crown piece below and internal to the left nipple, where he several times did not respond. Pricked with a pin even sharply, he says that it feels like the point of a lead pencil. When shown that it was a needle he was much surprised. Tested with test-tubes of hot and iced water, there was considerable delay in responding to the hot water, but he always said it was hot. Tested with ice water, he responded quicker and said it was hot and burnt him. He never once said it was cold. In fact, given to him with his eyes open to grasp in his hand, he said it burnt him, and he could not be induced to take it again. There is no loss of joint sensation and no loss of sense of position. There is well-marked, old optic atrophy of the right eye, some impairment of the sense of smell, none of taste. His mental condition indicates considerable dementia, he thinks that it is April, he does not know how long he has been here. He can give a pretty clear account of his previous life, which must have been a dissolute one.

Case 71.—*Tabes, osteo-arthropathy, drink, syphilitic history, hemiplegic attack, mania, dementia, mental improvement, pronounced signs of tabes.* (Reported by W. F. Forshaw, M.R.C.S., L.R.C.P.)

C. W., admitted to Claybury Asylum, July, 1900. Age on admission, 41. Occupation, ironer. Married, but has no children; has had no miscarriages. Shortly after admission was diagnosed as post-hemiplegic mania.

Resumé of patient's history previous to admission (her own account of it).—Had her left arm "pulled out when a girl." Has always been subject to ulcerated throats. Hair used to come out in combfuls. Barmaid for five years, addicted to drink (beer, whisky). Slipped down in street and fractured right tibia. "She was only aware that the leg was broken when she looked down at it and saw the bone sticking up." It was therefore painless, and indicated tabetic bone lesion. Had a seizure in street one year previous to admission, became unconscious and paralysed on right side after it. Attended Brompton for phthisis, and was advised to go abroad.

Mental state on admission (July, 1900).—She was very excitable and restless, wished she was dead, said "she was tired of life." She was dangerous to others, and threatened to kill the workhouse officials because they had been making irritating charges against her, and because she had been starved and illtreated. She soon, however, became depressed and despondent, and could not or would not give a rational account of her past life.

Physical condition on admission.—She was unable to walk, and had a right-sided hemiplegia. Her knee-jerks were not obtainable, and her pupils did not react to light or accommodation. She had no physical disease of the lungs or heart. Her right knee was much enlarged and leg everted; the patella was on the outer side of the joint, her foot was at a right angle outwards due to the ill-setting of an old fracture of the tibia. She was generally impaired in health and bodily condition.

A fortnight after admission her temperature suddenly rose to 103·8°, and she was unable to speak. No physical signs of illness found in any of the viscera; three days after this sudden onset of fever, she began to have seizures chiefly affecting the right side, numbering thirty-one in all. Ten days after cessation of seizures she was well again as regarded her speech, &c. She gradually improved physically, and two months later she was able to walk. She became quieter mentally, though had frequent maniacal outbreaks, and assisted in the ward work, and has remained in this state up to the present time, which will now be described in detail.

Present condition (March, 1902): *Mental.*—She is quiet and orderly, answers questions directly and correctly, has a good memory for remote and recent events, and is very anxious to get out of bed to help with the work. *Physical.*—There are no signs of hemiplegia now; her right leg is practically a "flail leg," it does not touch the ground when she is standing straight, quite two

inches shorter than the other. This shortening is at the knee joint and in the leg itself. The right knee-joint is swollen and full of fluid (fluctuation to be had easily, also the patella tap). There is great disorganisation, the patella is on the outside of the joint, there is a loose body in the joint just above the patella, which feels like a piece of bone probably broken off from the external condyle; crepitus is to be had easily by rubbing it against the patella; the internal condyle of the femur is very much expanded, especially antero-posteriorly, the joint is much wider than the corresponding one. This swelling came on suddenly, and reached its present size in three days; it is painless, and the skin is not abnormally heated. Her foot is more everted than it was on admission owing to the condition of the knee, and her leg moves abnormally in extension, flexion and rotation. Other joints of body appear normal. Her pupils and knee-jerks remain as on admission. She had an attack of acute shooting pains through the right side of her abdomen three weeks ago, "like knives running into me," and vomited in consequence. The sensation of her legs is very much changed. This change is most marked on the left leg, especially in the region supplied by the fifth lumbar root; she was anæsthetic to light touch over this region, could not discriminate between prick or touch, or hot or cold. Something similar on right leg, but not so marked; could distinguish between hot and cold. Sensations very faulty generally over both legs. There is no analgesia of thorax, but there is a well-defined belt of trunk light tactile anæsthesia extending from the lower border of the third rib to the lower border of the sixth rib, both front and back. Heart and lungs appear normal. She has had difficulty with her urine lately, and three days ago she had to be catheterised, 45 ozs. drawn off. Since then she has passed urine regularly, averaging between 60 and 70 ozs. per diem. She has a median scar on palate, it looks like an old cleft-palate operation. She speaks with a lisp.

A week later, April 4, 1902.—Patient has been in bed for the last ten days. The swelling of the right knee is much less marked; there is no heat or redness. General improvement since.

Interesting Hospital Cases.

Case 72.—*Macropsy and micropsy, transitory aphasia and right hemiplegia, doubtful diagnosis of general paralysis or syphilitic brain disease.*

H. B., aged 43. Came to Charing Cross Hospital for weakness in the legs. History of syphilis from the doctor. Pupils of

medium size, sluggish reaction to light. Knee-jerks present both sides. After leaving the hospital he was affected with a transitory aphasia and a right hemiplegia. Rigidity in his right arm and leg set in and persisted. He did not lose consciousness, but he suffered for some time with a headache; later on he was affected in the following way: whilst sitting at home he complained to his wife that the picture frames were empty, his trousers were moving up his legs, and that the handkerchiefs on the table were moving towards him. He could not sleep, and said the bed was being pulled away from him, and the sheets were being plaited. This was the first time his wife noticed anything wrong mentally. On April 28, on taking a walk with his wife he suddenly became rigid and drawn to his right side, nearly causing him to fall. Patient says that two years ago he had tingling sensations in his right foot, which he afterwards felt in his left foot. For the last six months he has had some difficulty in getting from his bed to the floor, having lost sensation in his feet. He has lost three stone in weight during the last year. There is nothing else noteworthy in his past personal history, except for the fact that for the past fifteen months, according to his wife, patient has dragged his right leg in walking.

Present condition.—Patient is pale, has a drowsy apathetic expression. He answers questions slowly, and although he is fairly accurate in his answers, it appears to cause him trouble in comprehending what is said, and in recollecting in order to answer. Both pupils equal, react sluggishly to light. Discs: nothing definitely abnormal discovered by Mr. Collins. Patient says his eyesight is becoming worse, and he complains that things appear to move, and that objects sometimes appear too large, and sometimes too small.

Case 73.—*One of a family of six suffering with Friedreich's disease. Pains, but no loss or diminution of cutaneous sensibility. Marked ataxy.*

A. W., aged 37, admitted to St. Pancras Infirmary, October 19, 1900. She is the eldest of six members of her family, all affected with Friedreich's disease. She was an out-patient under Dr. Ormerod for twenty-one years.

Family history.—On the father's side there is a history of fits, on the mother's side, grandfather paralysed. The illness began when she was 15 with jerky movements which the doctor thought was chorea. She was able to walk up to the age of 31, since then

her speech became affected, her back more curved, and the muscular weakness more pronounced. She is very feeble, being hardly able to raise the legs from the bed or to sit up, but able to use her hands and knit, but she would be unable to do this in the dark.

Present state.—Patient has a rather weak, fatuous, but mobile expression, there is more paresis on the right side of the face than on the left, the tongue is protruded slightly to the right. Her speech is thick, somewhat indistinct, slurred, lisping, and occasionally stumbling, but she is able to pronounce all the letters, and there does not appear to be any more difficulty with one word more than another. There is no tremor in the lips or tongue, and I could detect no nystagmus, although there were irregular, oscillating movements of the neck. The grasp is very feeble, and there is a difficulty and a slowness in unclasping the fingers, so that the hand seems to stick. She had suffered much with shooting pains, and they cause her legs to jump a great deal. There is marked ataxy and loss of sense of position in the hands, but she knows what I am doing when I straighten or bend the fingers, showing that she can appreciate joint sensation. In the lower limbs there is marked talipes equino varus, the muscles are somewhat wasted, the deep reflexes lost, the superficial plantars exaggerated. There is but moderate hypotonus. Sensibility to pain and touch good and correct in localisation, but the response is slow; but this may be only due to slow mental reaction, which is very marked when you converse with her. The epigastric reflexes on the left side lost, but slight crossed reflex of the right; right side absent, no crossed reflex. There is no anæsthesia or analgesia anywhere in the trunk, there is very marked scoliosis of the spine, which is due to a dorso-lateral curvature to the right. There is great weakness of the trunk muscles and of the erectores spinæ. She is quite clean in her habits, careful and intelligent, and in good bodily health, save the paralysis.

Case 74.—*Examples of optic atrophy affecting one child, facial paralysis another, and several others suffering with fits. No signs of syphilis on the body, but a certain history of syphilitic infection of the mother.*

This family history is of interest, because, had I not been able to obtain full particulars from the practitioner, I could not have found either from the mother or the examination of the children, that syphilis was the cause of the optic atrophy in the child who was brought to see me on account of blindness.

F. C., aged 11, was brought to me by Dr. Atkin suffering with blindness of both eyes since he was 7 years of age. The right eye became first affected, and then the left. He attended Moorfields for four years without benefit for optic atrophy, cycloplegia, and iridoplegia : on the card it is put, (?) Syphilis congenital. There are no signs of congenital syphilis now on the body, although Dr. Atkin informs me that he treated the boy when an infant for snuffles and a rash. Up to nine months ago he could see to play with other children, but he has gradually become quite blind, and has been sent to a blind school. There is slight evidence of facial paresis of right side, and the tongue protrudes towards the right. Grasp is good in both hands, and there is no loss of power. All deep reflexes somewhat exaggerated. Mentally he is intelligent, quick witted, and comprehends well.

History of maternity.—Mother married eighteen years, six children, and one miscarriage. Such a history with no signs of syphilis on the bodies of the children would allow the statement, no history of syphilis ; in fact, some authorities would cite this case as one, proving that optic atrophy could arise independently of syphilis ; perhaps it can, but this and many other cases that are cited do not prove it.

Miscarriage, seven months ; (1) Boy, died in a fit, aged 1 year and 9 months ; (2) Girl, now healthy, aged 15 ; (3) Girl, now healthy, aged 13 ; (4) Boy, the patient, aged 11, optic atrophy ; (5) Boy, aged 9, left facial paralysis at six months, commencing optic atrophy ; (6) Boy, aged 6, fits until five years old.

Dr. Atkins informed me that the mother, three months after marriage, suffered with sore throat, ulceration of the womb, and a characteristic rash for which he treated her.

Morbid Anatomy and Pathology.

Introduction.

We have already seen that the clinical cases show all grades of tabes with slight mental symptoms and pronounced cord symptoms to those of very pronounced mental symptoms and slight cord symptoms. Consequently there must be a number of intermediate cases between the two extremes, and it would be extremely difficult to decide whether they should be called tabes or general paralysis, and the invention of the term tabo-paralysis is an indication of this clinical difficulty. In like manner, and as a matter of expediency (especially among private patients) such cases are at first called locomotor ataxy. There can be no hard and fast clinical line between the two diseases; etiologically I have shown that they are practically identical; are they pathologically so? This question can be studied best by an examination of a sufficient number of these intermediate cases. Nageotte asserts that he found changes in the posterior columns in two-thirds of all forms of cases of general paralysis, and he looks upon the diseases as pathologically identical. Fürstner, who has made a most careful study of this subject, does not agree with Nageotte that the spinal cord exhibits typical tabic lesions in anything like so large a proportion. He concludes that the spinal cord is invariably affected in some part of its structure in general paralysis; he states that there certainly occur cases where the spinal lesion is first; also a series of clinical symptoms corresponding with the anatomical findings.

Dr. Watson finds posterior column degeneration of exogenous systems in five out of eight cases of juvenile general paralysis.

If it be admitted that syphilis is the most important (if not essential) cause of both these two diseases, and very strong proofs now exist of the truth of this doctrine, which is daily becoming more widely accepted, then, just as we find that alcohol, lead, diseased maize, ergot, and other

poisons may produce in one individual a morbid process in the brain, cord or peripheral nerves, while the disease process attacking the nervous system is essentially the same, so I will endeavour to show that the effect of the syphilitic poison on the nervous system may in one person produce a degeneration of the spinal afferent protoneurons, in another of the optic nerves, or the cerebral cortex, and, according to the structure affected primarily, the disease is classified as spinal tabes, optic tabes, tabo-paralysis or general paralysis.

Here I will also take the opportunity of remarking that some of the most rapidly progressive cases of paralytic dementia have been those in which, simultaneously with the brain affection, there has been cord affection also. A case of juvenile paralysis, published in the first volume of the ARCHIVES OF NEUROLOGY, p. 278, died within four months of the onset of the disease; several other cases (68, 69) likewise show a rapid progress. The onset of the cerebral symptoms generally, but not necessarily, leads to a cessation of the morbid process affecting the spinal cord and *vice versâ ;* it may then be asked, is the disease one affecting the vital energy of the neurons of the whole nervous system ; and in the struggle for existence, do those die upon which the greatest amount of stress falls, aided by contributory precarious vascular supply, or some devitalising condition, especially hereditary defective durability? How can, however, the environment affect the decay of structure, unless there is a deficiency in the nutritive properties of the environment of the neurons?

We might assume, hypothetically, that there is some biochemical defect or subminimal deficiency in the blood, the lymph, or the cerebro-spinal fluid, by which nervous metabolism may be rendered incomplete. Those nerve structures with the highest vitality, in a struggle for existence, would be able thus to extract a sufficiency of the necessary complex phosphorised materials for maintaining physiological equilibrium and for the formation of protagon ; whereas, if there is a total insufficiency, those with less *vis propria* would first waste and undergo atrophy. A vicious circle is very apt to

be established when structures presiding over important functions of the body are diseased, and then secondary complications arise and may be mistaken for the initial cause. I look upon these diseases as primarily a premature process of decay; the term "abiotrophy" recently introduced by Sir William Gowers (*Lancet*, May, 1902) expresses aptly in one word what has been said above, and previously expressed in my Croonian Lectures.

The disease, whether it is elective (affecting only some particular system of afferent fibres in the spinal cord or the optic nerves), or whether it affects only the cerebral cortex, is a veritable tabes or wasting. This wasting, however, may be general and affect the whole central and peripheral nervous systems. In many cases of tabo-paralysis the cord is so wasted that in a big muscular man, *e.g.*, Br—, 54, and Case 31, the cord was not so large as that of a two-year-old child. Both gray and white matter were atrophied, and without any proportional overgrowth of glia or thickening of membranes. The process as thus seen appears to be a biochemical failure on the part of the neurons to maintain physiological metabolic equilibrium.

The paroxysmal attacks of pains and crises, maniacal and epileptiform seizures, are clinical expressions of an irritant action, and suggest the formation or accumulation in the blood or lymph of some toxin which has a local action, unless we assume that the decay of the neurons (terminating in death) is accompanied by increased irritability. In the discussion of the changes in the brain, we shall see that there is much more reason to suspect the existence of an irritant toxin which produces acute destruction of the neurons and formative proliferation in the vessel walls and perivascular lymphathic sheaths, also active glia proliferation —all being the results of a conspiracy of morphological and biochemical factors. In the case of the spinal cord, the thickening of membranes and the changes in vessel walls are inconstant, and can in no way account for the elective destruction of fibres in the posterior columns; although as I have previously said, initial affection of certain segments of the spinal cord, as shown by the symptoms—especially the

cutaneous sensory disturbances—may be explained by a precarious vascular supply in these regions; but then, this is not the essential factor, only the contributory one. Morbid anatomy and clinical observation show that there is both systemic and segmental election in the pathological process.

Methods of Observation.

The tissues were obtained in a fresh condition and hardened in a manner suitable for staining by Nissl, Pal, Weigert, Marchi, and Marchi-Pal methods. Various other methods were employed (when necessary), such as the Heidenhain, Gudden carmine, and Bolton's iron alum; the sections were either cut in paraffin by the large Cambridge rocking microtome for the Nissl and Heidenhain methods and always of uniform thickness; or, for the other methods, cut in celloidin after hardening in Müller or Formol-Müller. A systematic examination was generally made of the spinal cord and roots at every level, together with the cauda equina. A number of the posterior spinal ganglia were also examined. Sections of the brain cortex in various situations, usually of Broca, first frontal, ascending frontal and ascending parietal, prefrontal, occipital, temporal and angular were made in a number of cases by Nissl method as well as by one of the before-mentioned methods to demonstrate the fibres. The medulla and, in a few instances, the pons and internal capsule were also examined; likewise the optic nerves and in some instances the retina, the peripheral nerves of the skin and muscles in a few instances, and in a considerable number of cases, the ulnar, median and sciatic nerves. I do not lay claim to have examined all these tissues in so systematic a manner as I have the cord and cerebral cortex; the observations have been made rather with a view of establishing the fact that the disease is one primarily of the central nervous system, and that the peripheral structures are affected later in the disease.

The spinal cord lesions of tabes and tabo-paralysis as regards the affection of the posterior roots and posterior columns are, as a rule, identical. In both diseases there is the same system of exogenous fibres affected in the same

situations in the spinal cord. The morbid process elects first certain groups of fibres and spares others, but eventually, in advanced cases, destroys nearly all the exogenous fibres. Many symptoms and signs, and especially the distribution of anæsthesia in the cases of tabes and tabo-paralysis (in which the cutaneous sensibility could be ascertained), indicated that certain segmental regions of the spinal cord are earliest affected; and systematic microscopic examination of the spinal roots and of the spinal segments showed that certain regions are more affected than others—viz., lower lumbar and sacral, mid, upper dorsal and lowest cervical. With few exceptions all the spinal cords were obtained from well-defined tabo-paralytic cases; but in some of those dying within the last year, I have systematically examined the patients for cutaneous anæsthesia, and made charts of the same, and correlated the results (Cases 31 and 46) with the microscopic investigation. Charts of the spinal degeneration at various levels have been made by means of Edinger's projection apparatus, so that the distribution of the degeneration can be studied at a glance.

Posterior roots.—Sections of the cauda equina in most of the cases were made by tying all the roots together, and embedding in celloidin. Microscopic examination almost invariably showed (whether the case was early or late) that degeneration or disappearance of fibres had taken place, relatively proportional to the atrophy of the exogenous system, in the lumbo-sacral region of the cord. Sections, longitudinal and transverse, which had been stained by Marchi fluid very seldom showed any degenerated black fibres, and they presented none of the appearances of Wallerian degeneration of nerves. When stained by Pal or Weigert, some fibres which stained blue might be normal in appearance, others appeared very much attenuated, as if the myelin sheath had atrophied; moreover, they stained a fainter blue owing to the thinning of the myelin. The bundles of posterior roots, which are normally considerably larger than the anterior roots, were much smaller and diminished in size, in proportion to the outfall of fibres. The interstitial tissue was sometimes increased, although there was not always a proliferation; the

vessels were often seen engorged with blood, but not more so than those of the undegenerated anterior roots. The walls of the arteries and arterioles were more often normal than not; when thickened, it was generally in a subject over 50, and the thickening was not limited to the posterior roots, but affected also the arteries of the comparatively healthy anterior roots. I do not remember seeing one which presented the typical character of recent syphilitic arteritis, though it is not uncommon to find a condition of arteriosclerosis, which might, of course, be indirectly of syphilitic origin.

Teased preparations of the posterior roots were made and stained with Marchi, with logwood and ammonium picrate, and with various other stains. The appearance presented reminded me of the condition found in a nerve after section, when all the products of degeneration have been absorbed. Empty neurilemmal sheaths with proliferated nuclei, vessels and connective tissue are the only structures found, as a rule, in the roots which have undergone degenerative atrophy. Some fibres still possessing a myelin sheath may be found amidst this tissue, if the root-fibres are not completely destroyed (*vide* fig. 38). The myelin does not show black globules like a degenerated nerve-fibre, but occasionally a fine black dust scattered through its substance, as if a slow molecular Wallerian change was taking place; or the myelin might be simply diminished in amount, and attenuated to various degrees in some places, while of fair thickness in others. Where the myelin is diminished, several nuclei of the neurilemmal sheath could be seen, as if the atrophy of the myelin had been followed by, or associated with, nuclear proliferation. Phagocytes containing blackened myelin were seldom seen, except in some few cases of rapid tabo-paralysis.

The roots in a case of ataxic paraplegia, where there was marked degeneration of all the long tracts of the spinal cord, presented quite a different appearance; for the myelin sheath was of normal thickness, showed nodes of Ranvier, and a very little interstitial tissue, and only the normal number of nuclei in the neurilemma. Some of the fibres

showed an appearance like Wallerian degeneration, but the majority showed no change; the morbid process was therefore intraspinal, whereas the degeneration of the sensory posterior spinal neurons of tabes is intradural, for the degen-

FIG. 38.

Two fibres from posterior root, eleventh dorsal, Case 49. Magnification, 400.

erative atrophy extends back as far as the spinal ganglion. My results, therefore, conform in great measure to the observations of Philippe upon this point. Before dealing more in detail with the distribution of the intraspinal degen-

eration of the exogenous system of the posterior columns, and the additional degeneration of other systems which may occur, we shall consider the changes which may be observed in the spinal ganglion itself.

Spinal ganglia.—The attractive theory of Marie based upon the neuron doctrine that the degeneration is due to a nutritional change in the posterior spinal ganglion cells is one which explains many facts, but the argument which is advanced against it is this: if it be so, why are not nutritional changes manifested histochemically by the Nissl method? Some authorities, Wollenberg, Ströbe, and others, have described changes in the posterior spinal ganglion cells; on the other hand, numerous observers have asserted that the changes which have been found were insufficient to account for the changes in the roots and cord. The examination of a large number of sections of posterior spinal ganglia, from the majority of the fatal cases I have recorded, has not satisfied me that the changes which I observed were sufficient to account for the disease. The changes in the cells were indeed insignificant as compared with the degenerative atrophy of the fibres emerging from the ganglion. In many instances, some few of the cells were shrunken, the nucleus eccentric and even the capsules empty, as if there had been a complete atrophic decay; but the majority appeared fairly normal, both the large, medium, and small sized. Frequently excess of pigment was seen, but the same might be found in the cells from any old person. Sometimes the great majority of cells of a ganglion stained blue by Pal, and by examination with an oil immersion, it was found that the protoplasm was beset throughout with fine dust like blue particles. The same may be seen in early fatty degeneration of muscle when stained by this method, and it may be that the protoplasm of the cells have undergone a recent fatty degenerative change. Stained with Marchi the same cells for the same reason may appear black. The process to my mind is acute, and may well be due to complications occurring in the tabo-paralytic towards the end of life.

Sections of posterior roots in animals did not, I find,

give rise to any degeneration of the spinal ganglion cells. Lenhossek at first thought there was a change, but further observations caused him to renounce this opinion. If neurilemmal cells play an important part in tabes, why are posterior roots invariably degenerated back to the ganglion?

Peripheral nerves—white rami, gray rami, sympathetic.—In a number of cases the peripheral nerves were examined, median, ulnar, and sciatic, sometimes the nerves of the skin, sometimes the nerves entering the muscles, *e.g.*, vastus internus. The white rami, gray rami, and sympathetic were also examined in a few cases, but a more systematic examination is now being carried on by Dr. Goldschmidt.

It may be stated generally that the peripheral cerebro-spinal nerves like the spinal cord of advanced cases of tabo-paralysis were smaller in trans-section than normal, and without any microscopic examination it would be said that they were apparently atrophied.

A section longitudinally through the spinal ganglion with a portion of the roots and issuing nerve attached stained by Weigert method, and examined microscopically, leaves no doubt in the mind of the observer that the disease is essentially one of the intradural portion of the spinal afferent neuron. He will observe the anterior root, consisting of normal fibres, lying by the side of the degenerated and completely atrophied posterior root (fig. 39), and he will conclude that no mechanical process of strangulation at the point of exit from the dural sheath will explain this fact. At the proximal end of the ganglion he will see very few fibres, and these much attenuated; whereas, at the distal end he will observe the fibres emerging of normal appearance, and as well stained as the fibres of the anterior roots. Again, he will consider that it is impossible for a vascular change within the ganglion to account for the distal portions of the T-shaped processes of the cells to have escaped.

I have, however, noticed in longitudinal sections of the posterior spinal ganglion with a long attachment of the ventral and dorsal roots, that the myelin sheath of fibres, which still possess the property of staining blue by the Pal

or Weigert staining, becomes sometimes more attenuated and fainter as one proceeds away from the ganglion towards the cord. Again, one may find the peripheral fibres proceeding from the ganglion quite healthy in appearance (as regards myelin staining) in an advanced case of tabes, and yet the remote sensory fibres degenerated. Dr. Anderson has kindly allowed me to quote some valuable observations which he has made upon the development of the myelin sheath in the posterior roots and peripheral nerves bearing upon this point, to be described later.

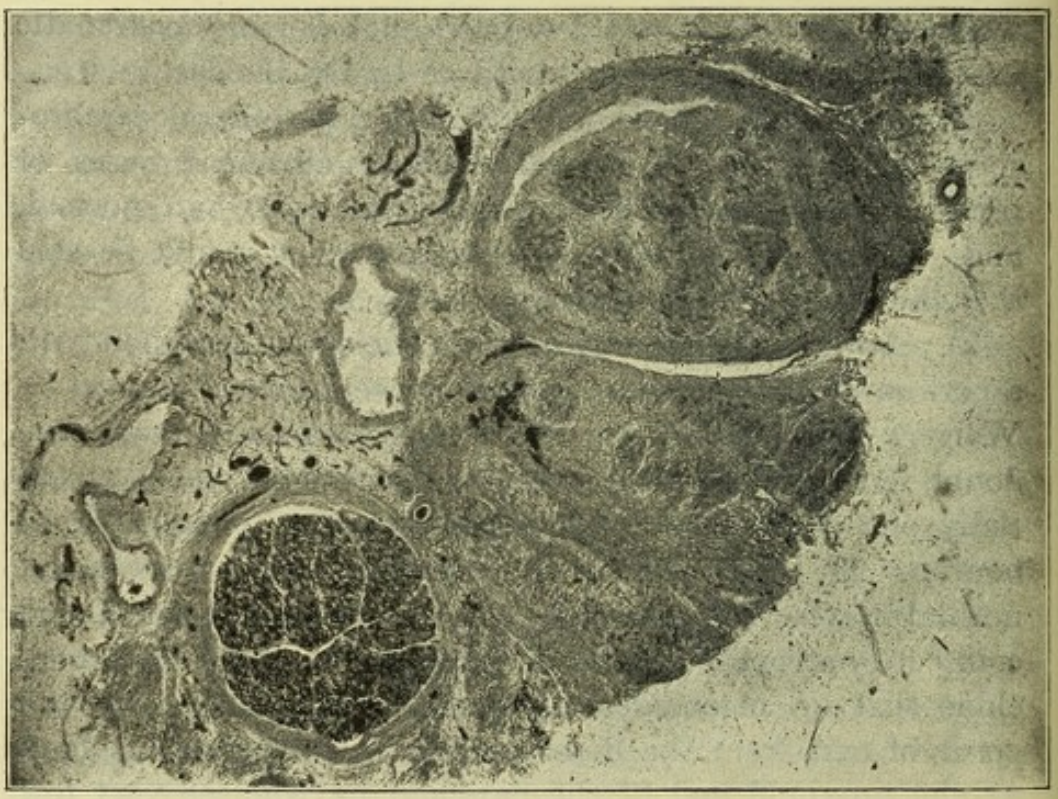

FIG. 39.

Photomicrograph of a transverse section of the posterior root (third lumbar). The small, dark round bundle of fibres is the undegenerated anterior root, the large sclerosed root is the posterior. Section stained by Weigert hæmatoxylin. Magnification, 20.

Examination of the Peripheral Nerves.

Transverse and longitudinal sections in some advanced cases showed changes, but in no degree sufficient to account for the symptoms, or in any way proportional to the atrophy of the posterior roots. The ulnar nerves of Case 31 showed

recent degenerative changes; some of the fasciculi of fibres contained a few black stained fibres by the Marchi method, due, apparently, to Wallerian degeneration; and in some places there seemed to be an excess of interstitial tissue and a diminution of fibres. In Case 49, however, where there was extensive muscular wasting of the small muscles of the hands with reaction of degeneration, and atrophy of the anterior horn cells and anterior roots, there was naked-eye degenerative sclerosis of the median and ulnar nerves. This was quite apparent when the nerves (after hardening in Müller) were cut in transverse sections, and showed a marked atrophy of fibres with substitution fibrosis. The sciatic nerve likewise showed a similar change; but there was not the same atrophy of the anterior horn cells, as seen in the cervico-dorsal region; the cells in the lumbo-sacral region, on one side more than the other, showed a process of regressive atrophy; the cells were there, but their processes were broken off, and there was not the same degeneration of the anterior roots. The process was very probably the same, but not so advanced, as in the cervico-dorsal region; still the outlying portions of the neurons had undergone atrophy.

The cause of this degenerative atrophy will be further discussed later; also, considerations will be advanced as to why the central projections of the posterior spinal neurons should be degenerated when the distal remain unaffected; it is generally admitted that the peripheral terminations of the nerves in skin and muscles are more likely to show degenerative atrophy than the portions nearer the cells of origin. My examinations of the skin are not sufficiently numerous, nor do I think the methods usually employed are sufficiently reliable, to make any definite statements with regard to the importance of the changes; yet the examinations I have made were sufficient to convince me that in early cases there was little or no appreciable degeneration, while in the late cases, although there was undoubtedly atrophy and disappearance of fibres, the changes were of far less importance as compared with changes in the posterior roots. This coincides with the obvious segmental root distribution of sensory disturbances observed during life.

The white rami were examined in a few cases in serial section; it was observed that there were some large medullated fibres, which could be traced into the sympathetic trunk, and a large number of small medullated fibres, together with some gray sympathetic fibres.

The Exogenous Systems of the Posterior Columns.

In all the twenty-eight cases examined, the three sets of coarse fibres entering the cornu-radicular zone, and proceeding respectively to the root zone of Charcot, to the cells of Clarke's column, and to form Goll's column, were affected; not always, however, to the same extent, for the relative degree of degeneration of each of these three systems of fibres depends upon the relative degree of degeneration of particular roots. For, although every root in the lumbo-sacral region, where the morbid process in the great majority of cases commences, contains fibres belonging to each of the three systems, namely, spinal, cerebellar, and cerebral, yet some roots, according to the functions of the structures innervated by them, contain many more cerebral afferent fibres than cerebellar and *vice versâ*. The appearance of Clarke's column at the level of the second lumbar segment and its disappearance at the second dorsal is an indication of the important relation of this system of neurons to the muscles of the trunk and the muscles used in maintaining the static position.

I have shown[1] experimentally that the very large first sacral posterior root of the monkey possesses a large number of fibres belonging to all three systems, but especially to the cerebral system; this root is distributed to the whole of the sole of the foot and its deep structures; it is, therefore, of great importance in conveying sensory impulses essential for the maintainance of gait and station.

Examination by the Marchi method of the different segments of the cord, after section of this posterior root in the monkey on one side, showed: (1) That a very large number of fibres of this root enter into the formation of the

[1] "Die Zuführenden Kleinhirn Bahnen des Rückenmarks bei den Affen." F. W. Mott. *Monatschrift fur Psychiatrie*, vol. i.

column of Goll, apparently nearly half of the fibres of this tract in the cervical region being degenerated, and the degeneration uniformly distributed over the whole triangle; (2) that a considerable number of fibres enter the root zone of Charcot, in the first sacral segment especially, but also in the segments above and below; (3) that at the third lumbar segment the only degenerated fibres found in the posterior column consist of a band which extends from the mid line back to the periphery, leaving internal to it a small triangle undegenerated, probably tail fibres.

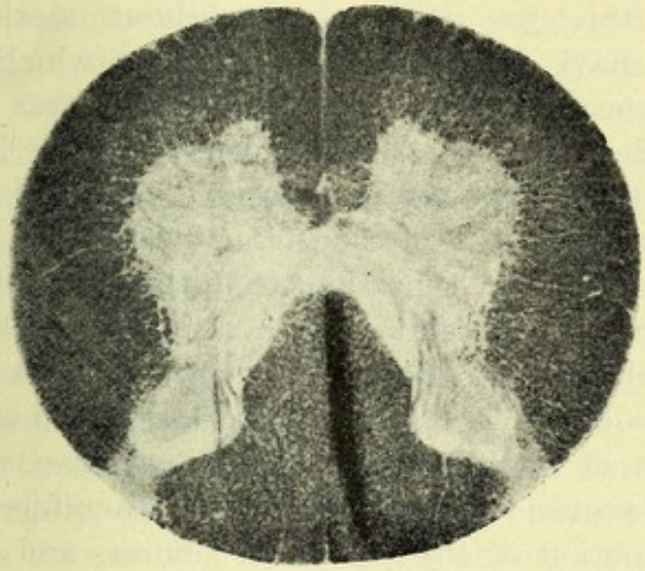

FIG. 40.

Photomicrograph of section (third to fourth lumbar segment) of spinal cord of monkey, after section of first sacral on one side. The black band seen represents the degenerated fibres belonging to the cerebellar and cerebral systems. It will be observed how very extensive this degeneration is. As soon as Clarke's column appears at the first lumbar a large number of these black fibres run forward to end in a plexus around the cells, leaving the fibres of Goll's column as a compact triangle posteriorly.

As soon as Clarke's column begins to appear at the upper part of the second lumbar segment, the anterior half of these degenerated fibres runs forward to end around its cells; and not until about the eleventh dorsal segment do these degenerated fibres cease to be found entering Clarke's column. The degenerated fibres of Goll's column then form a definite posterior internal triangle, which can be followed up to the medulla. This experimental evidence shows what

a very important part this root, which is so often affected in tabes, must play in the production of ataxy of gait and station, by the cutting off of guiding sensations to the cerebrum and cerebellum.[1]

The padded foot or complete anæsthesia of the sole, with abolition of cutaneous reflex, means, therefore, abolition of the fibres for cerebral and cerebellar impulses, and explains the increase of the ataxy when the sole of the foot is anæsthetised by cold. The ataxy, I am of the opinion, has a decided relationship to the atrophy of the plexus around the cells of Clarke's column, and I have not seen any case in which this was pronounced without marked ataxy, whereas I have seen other examples in which this was not pronounced, although Goll's column was markedly affected. Several cases showed comparatively little cutaneous anæsthesia, and yet there was very marked ataxy.

There is another set of exogenous fibres which, according to Bechterew, convey sensations from the skin ; these fibres are small in size and run a very short intramedullary course. In the lumbo-sacral region they occupy at least two-thirds of the zone of entry, or zone of Lissauer, and give origin to numbers of fine collaterals which arborise around the cells of the posterior horn. They do not, therefore, take part in the formation of the posterior column, and it may be presumed that these fibres convey sensory impressions from the skin. This is more easy to understand if we accept the hypothesis that the same fibres conduct pressure and pain impressions, but that quality and intensity of stimulus determine the sensation produced. In syringomyelia, however, we have sensory dissociation, touch being perceivable, but heat, cold, and pain not, for the reason that the grey matter is destroyed and the exogenous fibres of the posterior column are intact ; it may be presumed then, that the gray matter conducts painful sensations, and the posterior columns conduct tactile ; but the segments of gray matter of the posterior horn are united by long and short ascending

[1] Anderson has found that section of the seventh post-thoracic root (corresponding to the first sacral) produces in kittens an atrophy of the cells of Clarke's column, and he confirms the above observations of mine.

and descending association fibres, and their preservation, at any rate for a considerable time, may account for the delay, yet eventual transmission of sensory stimuli, in tabes, to the brain.

The fine fibres of Lissauer's tracts were in a few cases not affected at all. In many cases (eleven out of twenty-eight) when there was a marked affection of the root zone of Charcot, the fine fibres of Lissauer's tract were but slightly affected. In a few instances where it had been possible to investigate the sensory cutaneous disturbances during life, it was found that there was a relationship between the degree and extent of the same, and the degree and extent of distribution of atrophy of Lissauer's tract. Head and Campbell call attention to the great number of small cells in spinal ganglia of the trunk region as compared with the cervical and lumbo-sacral regions. They consider that the small cells subserve the function of pain, but some are no doubt afferent splanchnic neurons.

Several fatal cases were examined in which the sensory disturbances were either absent or comparatively slight, yet the motor incoordination was marked and the previously mentioned three sets of fibres in these cases were atrophied.

Occasionally, as Redlich and others have observed, in early cases terminating fatally acute changes may be found affecting the intramedullary portion of the exogenous neurons, and not the roots. Case 52 was an example of this, where the patient did not long survive the onset of the spinal symptoms, and we find a number of recent degenerated intramedullary fibres in the lumbo-sacral region, limited to definite systems (fig. 41). The coarse internal fibres entering the cornu radicular zone are seen degenerated at all levels of the lumbar and sacral regions; the black degeneration products are neither found in the entering zone of fine fibres of Lissauer's tract, nor in any of the sections of the posterior roots attached to the sections of the cord; the degeneration is therefore intramedullary and selective.

No cutaneous disturbance was observed during life, although there was ataxy; the coarse degenerated fibres belonged to (1) spinal reflex, (2) cerebellar, (3) cerebral

afferent systems, and occupied respectively (1) the root zone, terminating at the base of the posterior horn, (2) the fibres entering Clarke's column, (3) the fibres entering Goll's column.

There was no meningitis which could account for this intramedullary degeneration; moreover, if this were the cause, it should not be selective in sparing the fine fibres of Lissauer's tract. In such a chronic disease as tabes usually is, it is quite probable that a process of degeneration akin to

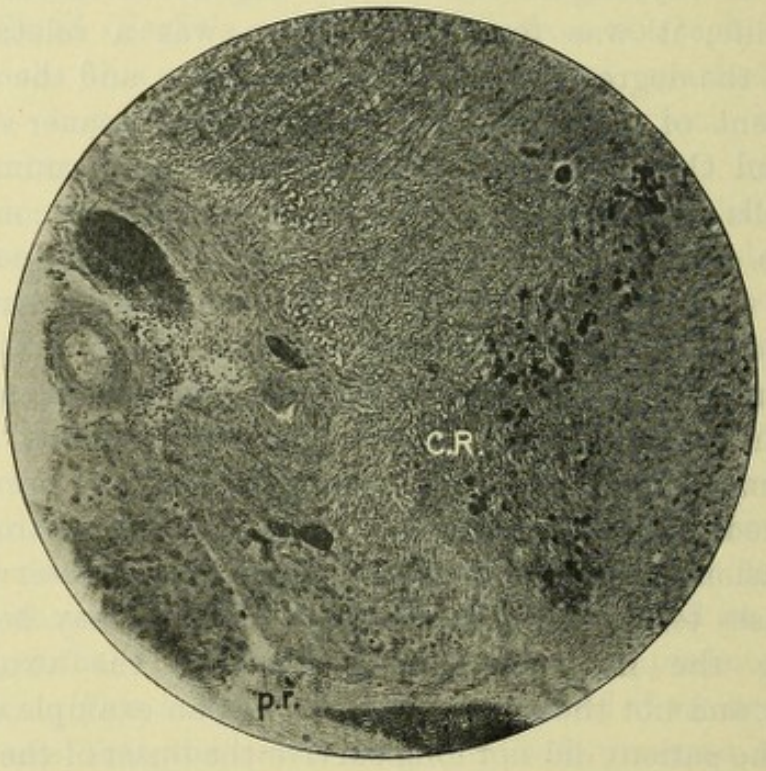

FIG. 41.

Section of spinal cord, first lumbar, Case 52, showing degenerated fibres in the cornu-radicular zone, C.R. The posterior roots are free from degeneration; so is Lissauer's tract.

Wallerian degeneration frequently occurs, the products being absorbed; but it is likely that all degrees of the process of myelin disintegration may take place; in some instances it is slow wasting, due to disintegration exceeding normal integration, and involving a gradual process of atrophy; in other instances, a rapid acute disintegration occurs akin to Wallerian degeneration, and between the two extremes there are probably all grades of destructive change.

The Endogenous Systems of Fibres of the Posterior Columns.

In the nine cases where there were very marked ataxic symptoms, the following results were obtained by microscopical examinations :—

(1) The descending endogenous system of fibres was markedly affected in six instances, and in three very markedly.

(2) The ascending endogenous system (cornu-commissural of Westphal) was never completely destroyed *throughout* the cord, even in the most advanced cases, although in one or two instances, *e.g.*, 49 and another case, notes of which are not recorded, but which is contained in the Table, it was almost absent in some regions.

In cases of endogenous system degeneration, there was invariably a very marked atrophy of the exogenous system. In nearly all the cases which commenced with optic atrophy, eventually terminating in blindness, there was no appreciable affection of the endogenous system; this, however, was not invariably the case, striking exceptions being Cases 28 and 49. There was a decided relationship between the degree of affection of the endogenous systems of the posterior columns and the degree of ataxy, moderate atrophy of the endogenous systems occurring in persons who were well on in the second stage of locomotor ataxy. All those with marked or very marked atrophy were in the advanced bed-ridden paralytic stage, and one or two presented muscular atrophy. All those in the pre-ataxic stage showed no degeneration of the endogenous systems, although there was extensive atrophy of root-fibres and their intramedullary projections in many instances.

Origin of the fibres of the endogenous systems of the posterior columns.—Many observers have shown that transverse lesions of the spinal cord produce descending degeneration of the posterior columns. Dejerine and Théohari point out in their communication that Schultz, Bruns, v. Lenhossek and Obersteiner consider that the comma tract is composed of exogenous fibres; Tooth, Pierre Marie, Gombault, and Philippe and Daxenberger, that it is composed of endogenous fibres. Dufour and Philippe (in his thesis)

think that the comma tract and the dorso-median degeneration in the lumbo-sacral region represent one and the same system of endogenous fibres. Purves Stuart in *Brain* has recently expressed the same opinion, based upon the examination of the descending degeneration resulting from a crush of the cervical region. Flatau, on the contrary, believes that for the greater part of its extent, the descending degeneration is of radicular origin. Hoche remarks that the long extent of the comma tract does not agree with what is known of the descending branch of the posterior roots; he regards the peripheral degenerated field and the comma tract as two independent systems. Dejerine and Théohari formulate the following conclusions, based upon an analysis of experiments upon animals, and upon the descending degenerations observed in the posterior columns in man, in consequence of pure radicular lesions, and upon a comparison of these degenerations with the descending degenerations observed in two cases of transverse lesions of the cord:—

(1) The degeneration in the comma tract of Schultze is due in part to the lesion of the descending branches of the posterior roots; the longest fibres of this tract, however, are of endogenous origin.

(2) The small anterior degenerated zone, which does not extend below the segment subjacent to the transverse lesion, represents short endogenous longitudinal commissural fibres.

(3) The peripheral bundle of Hoche, dorsal region (septo-marginal, Bruce) is continuous with the central oval area of Flechsig and the triangle of Gombault and Philippe, and represents a very long system of longitudinal commissural fibres; it is a system of endogenous fibres.

(4) The triangle of Gombault and Philippe contains (besides endogenous fibres) a great number of radicular fibres; it is therefore of mixed origin.

My own observations, experimental and otherwise, tend to support these views in great measure, although I am inclined to agree with Dufour, Philippe, and Purves Stuart that the descending fibres of endogenous origin belong to one system.

I consider that these fibres, like the exogenous systems

of fibres, are of long, medium, and short lengths, and serve for the correlation of sensory function of different segments of the spinal cord.

Observation of many cords, in some of which, in the mid and upper dorsal regions, there was a complete atrophy of the root-fibres in cases where during life there was a corresponding loss of cutaneous sensibility of the trunk, showed

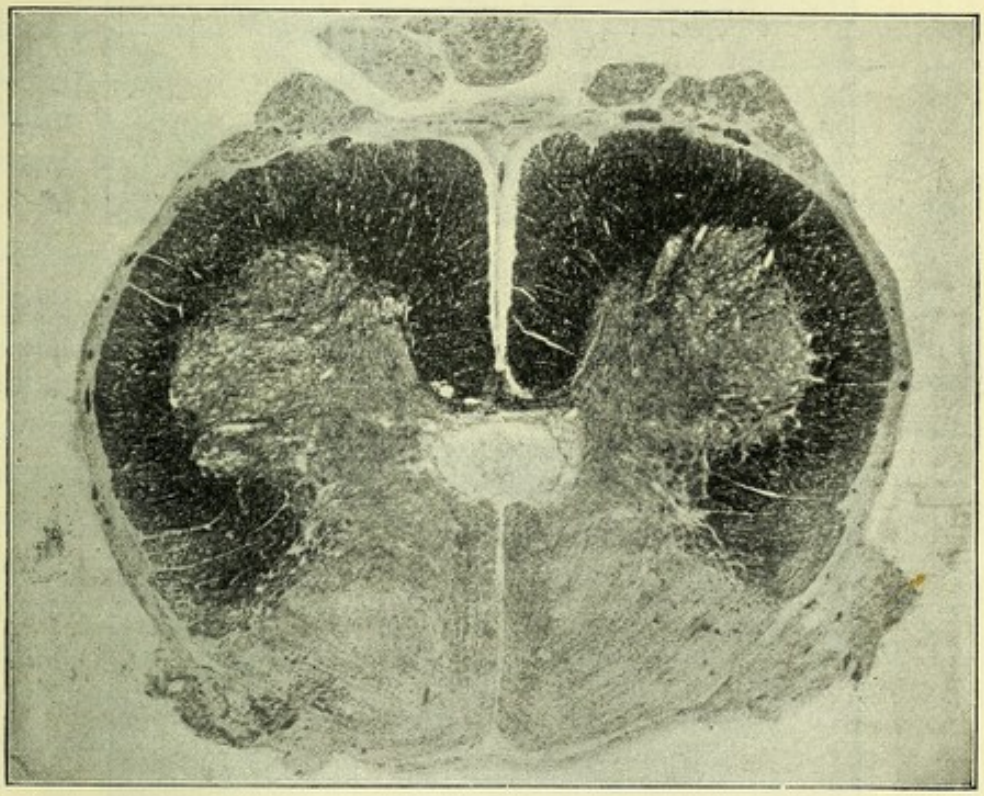

FIG. 42.

Photomicrograph, lumbo-sacral region, of an advanced case of ataxy (tabo-paralysis). The posterior columns are almost completely denuded of fibres, a few are left in the cornu-commissural tract and the oval area of Flechsig. There is also sclerosis in the crossed pyramidal tracts on both sides. There is not much shrinking of the posterior columns, and the meninges are not more thickened posteriorly than anteriorly. The central canal is dilated and filled up with glia tissue.

still a large number of fibres in the situation of the comma tract, and the posterior internal zone extending thence along the dorsal margin of the posterior columns, and continuous at the first lumbar with a small triangle at the side of the median fissure. The fibres in this triangle, as one proceeds to lower levels, are seen to extend up the median fissure,

and become continuous with the oval area of Flechsig in the lower lumbar and upper sacral regions, and this again is continuous with the median triangle of Philippe, situated in the lowest sacral segments, *vide* photomicrographs 42, 43, 44.

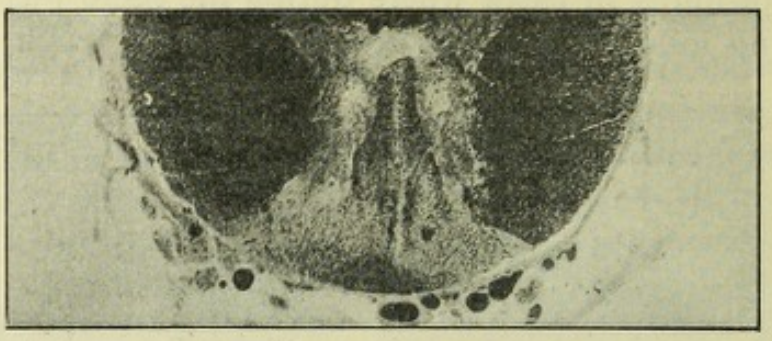

FIG. 43.

Photomicrograph of first lumbar segment (Case 31), showing cornu-commissural zone and posteriorly a small triangle of fibres in the mid-line of endogenous origin.

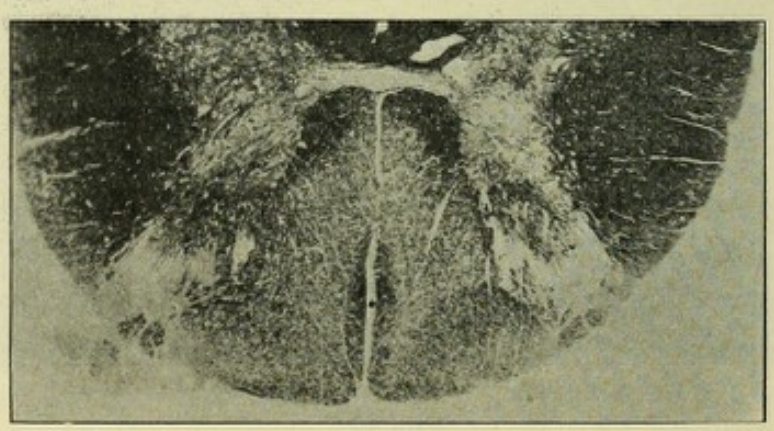

FIG. 44.

Photomicrograph of third lumbar segment (Case 52), showing degeneration of intraspinal portion of exogenous fibres of posterior columns; the ascending and descending endogenous fibres are intact. The continuity of the fibres of the posterior internal zone, the septo-marginal, and the oval area of Flechsig as one tract, is well shown.

In cases of apparently complete degeneration of the root-fibres in the mid-dorsal region, one finds a set of fibres giving off collaterals running on the inside as well as on the outside of the column of Clarke: (*a*) The inner set of fibres, continuous partly with fibres of the cornu-commissural zone of the same side, partly with fibres of the cornu-commissural

zone of the opposite side. (*b*) The outer set of fibres coming from the posterior horn and running forward to the base of the anterior horn, commingling there with the inner set.

This arrangement is especially well seen in Cases 39 and 46 (*vide* photomicrographs, p. 157 and Plate IV.).

Cajal has described cells in the middle of the posterior horn which give off fibres entering the posterior columns; these give rise to ascending endogenous systems of fibres in the cornu-commissural tracts, many of the fibres decussating to form the posterior commissure. The cells which give origin to the fibres of the descending system are possibly situated at the junction of the anterior and posterior horns.

In cases of complete root atrophy occurring in the lumbo-sacral, upper, and mid-dorsal regions as exhibited in Cases 39, 40, 45 and 49, numbers of very fine myelinated fibres can be seen in the substantia gelatinosa of the caput cornu posterioris; they can be seen when the fine fibres of Lissauer's tract are completely absent, they form a delicate plexus, and can be observed running horizontally across the base of the caput cornu posterioris. These are probably axis cylinders of the small sensory cells of this region. Again, in such cases of absence of root-fibres, numbers of fine collaterals can be seen in the gray matter opposite the root zone; it is probable that these are derived from the descending endogenous fibres.

In degenerative atrophy affecting the descending endogenous fibres the first to disappear are those in the dorsal region, occupying the comma-shaped tract, its dorsal club-shaped projection and peripheral extension towards the median septum; the next are the fibres of the oval area and posterior triangle of Philippe, with which they are continuous.

Degeneration of the Spinal Cord, other than the Exogenous and Endogenous Systems of the Posterior Columns in Tabo-Paralysis and Tabes.

In advanced cases of tabes sometimes there may be degeneration of the cells of Clarke's column, and as a result thereof, atrophy and sclerosis in the two afferent systems of fibres proceeding from these cells to the cerebellum, form-

ing respectively the ventral and dorsal cerebellar tracts (Case 40).

By far the most frequent and important associated degeneration and subsequent sclerosis is to be found in the pyramidal systems of fibres. In twenty out of twenty-eight asylum cases which I have examined there has been some degeneration in some part of the course of the pyramidal systems. This pyramidal degeneration is evidence of cortical affection, and is due either to chronic atrophic changes in the cortical psycho-motor neurons or acute destructive changes. Sometimes the two are associated.

Examination of the spinal cords of twenty-eight cases of tabo-paralysis has shown me that the affection of the pyramidal systems is in relative order of frequency as follows:—(1) *Primary degenerations.*—(*a*) Primary degeneration of both crossed pyramidal tracts, fairly equal on the two sides. (*b*) Degeneration of both pyramidal tracts, but more marked on one side than the other; this degeneration is slow in progress, not revealed as a rule by the Marchi method, but is shown by those methods which demonstrate sclerosis. Sclerosis is only revealed distinctly in the lumbo-sacral region in a situation corresponding with the position of the crossed pyramidal tract in that region; it can be followed as high as the mid-dorsal region in some cases, but becomes less and less discernible the higher we proceed in the cord. Such may be partly due to intermingling with healthy fibres, but I believe it is mainly due to the fact that this degeneration, which does not involve the direct pyramidal tracts, is occasioned by a slow progressive atrophic process affecting the cortical psycho-motor neurons with the longest axons, therefore those which have their terminal arborisations in the lowest regions of the spinal cord. The pyramidal fibres of the direct tract have a shorter course and consequently are not so remote from their seat of nutrition; as a rule, therefore, are not wasted.

Acute degeneration.—(*a*) Degeneration of the crossed pyramidal tract on one side, and direct on the other.[1]

[1] Starlinger has described two cases in which there was degeneration of the whole pyramidal tract from the cortex. The same was found by Boedeker and Juliusberger, *Neurol. Centralblatt*, No. 17, 1897.

Examined by the Marchi method one usually finds well-marked Wallerian degeneration, which may be traced up through the medulla, pons and capsule to the cortex, and is due to destructive changes in the cerebral cortex of one hemisphere; it is associated clinically with unilateral epileptiform seizures and hemiparesis or hemiplegia; sometimes with hemianæsthesia and hemianopsy, Cases 45, 54. (*b*) Bilateral, but usually unequal, degeneration of both crossed and both direct pyramidal tracts, which can be traced up to the cortex, associated clinically with severe bilateral epileptiform convulsions, and sometimes opisthotonus or emprosthotonus. The acute vascular changes in the cerebral cortex may be due to arterial thrombosis, brought about by syphilitic endarteritis, causing localised and general softening; this, however, is extremely rare. Occasionally it is an arteriosclerosis with multiple miliary softenings, and still more frequently examination of the brain shows vascular stasis, blocking of veins and capillaries, which may, in rare cases, proceed to thrombosis of the large veins opening into the longitudinal sinus, and cause red softening. The most frequent cause, however, is the characteristic chronic inflammatory affection of the cerebral cortex of typical general paralysis, with marked irritative cell proliferation of the capillaries and within the perivascular lymphatics. Where the degeneration of the pyramidal tract is unilateral, or more marked on one side than the other, there is invariably a greater wasting of the corresponding hemisphere, which contains the cells from which these fibres take their origin. In no case does the examination of the cortex show that the changes are solely of vascular origin; the atrophic process is partly due to chronic progressive atrophy, and partly due to acute degeneration. Thus we find the tangential fibres absent or greatly diminished in the prefrontal region of both hemispheres in cases where there is only unilateral pyramidal degeneration and unilateral fits. The cortical changes will be referred to in fuller detail later on. It may, however, be remarked that in all cases where there have been prolonged epileptiform seizures of recent, but not too recent, date, Wallerian degeneration in the pyramidal systems was found.

Each seizure, whether maniacal or epileptiform, connotes cerebral irritation, vascular reaction, with cell proliferation and acute neuronic destruction, consequently a series of seizures will be followed by a series of degenerations. The appearance presented, however, by the degenerated fibres is not quite like that produced by experimental lesions. A section of the pyramidal tract stained by Marchi method

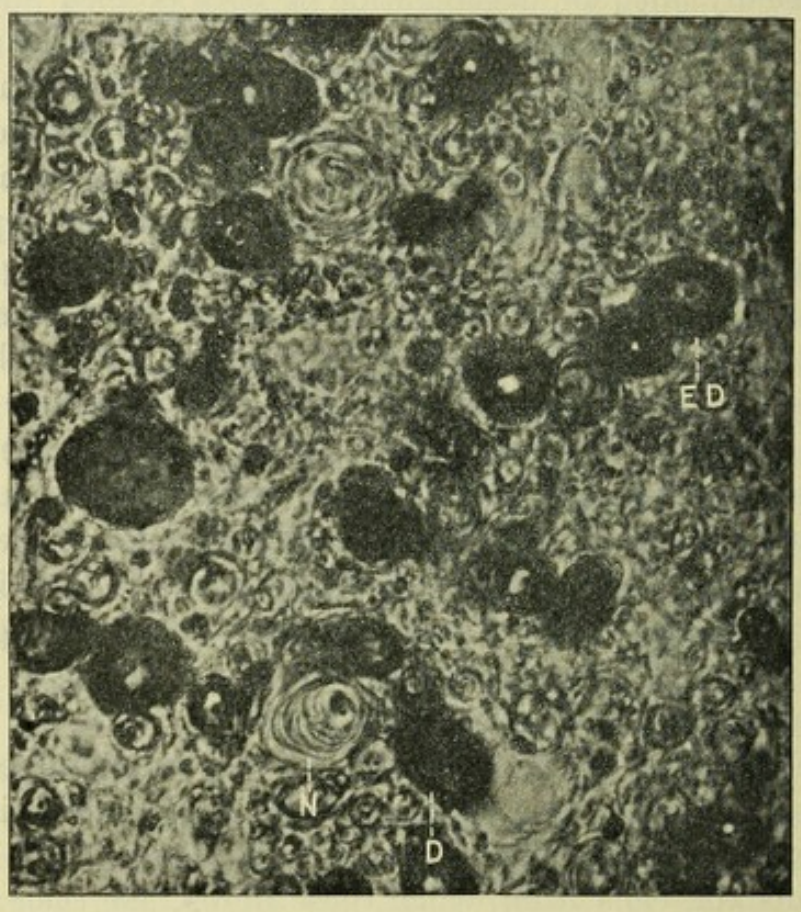

FIG. 45.

Photomicrograph of the right crossed pyramidal tract upper dorsal region of cord (Case 54). Marchi staining; magnification, 750. The smaller intervening undegenerated fibres are partly commissural ground fibres.

shows normal fibres, N; fibres in which the sheath stains black, but still retaining an axis cylinder, E D; others black throughout, indicating complete degeneration, D; lastly, empty spaces, indicating destruction of a fibre and absorption of the products. The smaller black degenerated fibres may be terminal branches of the pyramidal fibres.

In those cases of tabo-paralysis in which the disease is characterised principally by a slow progressive dementia

without epileptiform seizures, and in which microscopical examination shows a chronic atrophic degeneration of the neurons, affecting especially the molecular layer and the small and medium-sized pyramids with the corresponding atrophy of their association fibres, there is very seldom (and when present but very slight) Marchi reaction of degeneration; likewise there is no Marchi reaction of degeneration in the posterior columns in cases of chronic tabes dorsalis. Many of the spinal cords of these cases of tabo-paralysis were remarkably small, some not any larger than that of a child of two years (Cases 31, 52, 57), and this small size can only be explained by supposing that there is a general failure in the metabolic processes of nutrition of the nervous system. In tabes dorsalis the dwindling in size of the myelin sheath and its eventual disappearance, usually without any evidence of the Marchi reaction, makes one believe that there is a slow progressive process of failure in constructive metabolism of the neurons. The myelin sheath, together with the collaterals, which are the latest development of the neurons embryologically, are the first to suffer; and why in one case the brain, in another the cord, or in some both fail, can only be explained by the intervention of some contributory factors which determine a *locus minoris resistentiæ*, such as vascular supply, stress, and heredity.

Changes in the Anterior Horn Cells.

In advanced cases of tabes, various deformities arise in consequence of amyotrophy; the small muscles of the hand and foot are specially affected, also the muscles of the leg, particularly the peronei and dorsal flexors, giving rise to various forms of club foot. Electrical examination of the muscles usually shows no reaction of degeneration, but in rare instances, *e.g.*, Case 49, it does. Occasionally this may be due to chronic lead poisoning, as described in a case by Redlich, who claims that a typical posterior column degeneration of tabes was produced by lead poisoning, independent of syphilis; associated with it, however, was a chronic anterior polio-myelitis.

Some cases of progressive general paralysis apparently are

due to chronic lead poisoning, and the fact that a tabic[1] lesion of the cord can be produced by this cause, tends to support the unity of the two diseases.

It is only in advanced cases of tabes that one finds changes in anterior horn cells, and it is probable that these changes are due to total destruction of the sensory neurons which are in relationship with them. Warrington has shown that section of a large number of posterior roots produces chromolytic changes in these cells. I have been rather astonished at not finding more evidence of this in tabes and in the sections which I have prepared from cords of animals in which posterior roots have been cut. Possibly this was due to the fact that the animals were kept alive a much longer time than those of Warrington. Again, in cases of tabes, where there has been a very complete degeneration of the posterior roots, I have failed to find these chromolytic changes; but when such have been seen (besides the destruction of the posterior roots), there was a very marked atrophy of the endogenous fibres of the posterior columns. This atrophy prevents stimuli entering from other regions in which the roots have not been completely destroyed, and marked changes, indicating progressive nutritional failure and even, rarely, atrophy of the anterior horn cells may occur. A similar condition of the cells was observed in a case of very chronic rheumatoid arthritis, with muscular atrophy. All the muscles responded to electrical stimuli, but owing to the joint affection, a progressive change, presumably, took place in the anterior horn cells; their dendrons were broken off, the cytoplasm contained an excess of pigment and a diminution of the Nissl bodies.

In Case 49 of tabes, in which the change in the anterior horn cells was most pronounced, the cells exhibited the following characters. In the lumbo-sacral region, especially at the level of the first sacral, the anterior horns show the cells having their processes broken off; there is a perinuclear chromatolysis and marked excess of pigment. There is no marked proliferation of the intervening glia substance, as the

[1] "Tabes Dorsalis und chronische Blei-vergiftung." *Wiener Medicinische Wochenschrift*, 1897.

accompanying photomicrograph shows. This case was one of arm tabes, and many years before he died the notes state that there was complete analgesia and anæsthesia of the arms, a very unusual condition in tabes, as the anæsthesia very rarely extends to the distribution of the upper cervical roots. Then it was noticed that there was a progressive wasting in the small muscles of the hand, more marked on one side than the other, with eventually reaction of degeneration. Microscopic examination of the cord in the cervical regions showed an atrophic degeneration of the anterior horn cells in the upper two dorsal and last cervical segments, with a complete absence of fibres in the posterior roots and pos-

FIG. 46.

Case 49.—Amyotrophic tabes, anterior horn cells, first sacral segment showing chronic degenerative changes, breaking off of processes and excess of pigment. Magnification 300 diameters.

terior columns of the segments of the spinal cord which correspond to the origin of the fibres which enter into the formation of the brachial plexus. There was a considerable atrophy of fibres also in the upper cervical segments; probably, therefore, owing to the accompanying destruction of the ascending and descending endogenous system of fibres in the posterior columns, all direct and indirect reflex spinal stimulus to the anterior horn cells had been abolished for many years.

We can explain amyotrophic tabes in two ways: either the atrophy of the anterior horn cells has been brought about by a superadded chronic polio-myelitis, or the extreme tabic

atrophy of the sensory neurons, which are in relation with those segments, has given rise to a secondary trophic change from the abolition of the stimuli incidental to their vital activities, similar to that which occurs in the sensory neurons after amputation of a limb. This latter hypothesis seems probable, even though we found in the small muscles of the hand the very unusual condition of electrical changes; but then the affection was essentially the same as in the small muscles of the foot, only of longer duration. The microscopic changes met with in the first sacral and ulnar segments of the cord were essentially the same, although the latter was more advanced.

Why are the small muscles of the hand and foot so frequently affected by this amyotrophy, as compared with the large trunk and limb muscles? The latter may derive some reflex tonus by impulses *descending* from the cerebellum, partly incited by afferent impressions from the semicircular canals (*vide* experiments of Bickel and Ewald) partly by peripheral afferent impressions transmitted by the posterior roots through Clarke's column to the ventral and dorsal cerebellar tracts, which are cut off in tabes.

Again, we have seen that an important association system of descending endogenous fibres of varying lengths exists in the posterior columns, that it is seldom entirely destroyed (only in the most advanced cases), and that these fibres in the dorsal and upper lumbar region are brought into relation with the anterior horn-cells and the cells of Clarke's column at different levels, consequently stimulus may enter the cord in regions where roots are, in the vast majority of cases, still partially preserved. By means of these association fibres of the posterior columns distant neurons in Clarke's column and in the anterior horns may be affected, and thus preserve their nutritional and functional activity, and a certain amount of reflex spinal and reflex cerebellar tonus of the muscles. This is probable, seeing that the distribution of cutaneous anæsthesia and the microscopic examination of the roots and spinal segments of a large number of the cases which I have recorded indicate that the roots which longest maintain function and structure are the cer-

vical above the seventh, the lowest dorsal and upper lumbar; while the regions most likely to be affected are the lowest lumbar and upper sacral, the upper dorsal and the lowest cervical (*vide* figs. 3 and 4).

It was only in a case of arm tabes where there was during life evidence of affection of all the cervical roots by a complete anæsthesia of the arm that an amyotrophy in the upper limb was observed, although it was noticed several times in the foot. When it occurred in the foot there was always a very marked degree of analgesia and anæsthesia of the legs, indicating a very complete posterior root destruction in these regions. These synergic association systems subserve spinal co-ordination, and their degeneration plays an important part in the later stages of tabes.

Changes in the Optic Nerves.

In eight out of twenty-eight fatal cases (nearly 30 per cent.) there was optic atrophy with blindness. Microscopical examination of the optic nerves was not made in a good number of the cases, therefore exact statistics cannot be quoted, but probably 60 per cent. of the cases would show some degree of optic atrophy. The clinical history of some of the cases would suggest that the optic atrophy is not always primary, but may in some instances be due to syphilitic basic meningitis. Microscopic examination, however, does not help to decide. In several cases in which there was no definite naked-eye optic atrophy, one found a diffuse degeneration by Marchi method, also a large number of empty spaces as if fibres had disappeared. Case 54 was remarkable in the fact that the optic nerves were smaller than natural, yet microscopically showed no overgrowth of glia tissue. The component fasciculi of the nerves were small, as if there was a general atrophy as inexplicable as the small spinal cord so frequently met with. In several advanced cases the retina was examined (*vide* Case 65).

Developmental Defects.

In three cases (41, 46 and 61) out of the twenty-eight examined there was a true *heterotopia spinalis*. As I have

examined a very large number of spinal cords, normal and abnormal, but have never met with this condition except in these three cases of tabo-paralysis, it must be something more than coincidence that 10 per cent. of tabo-paralytics should have this defect; it supports the opinion of those who maintain that an hereditary deficiency may be an important causative factor in the production of the disease.

Morbid Anatomy of the Brain.

The changes in the brain in tabo-paralysis do not differ essentially from those of ordinary paralysis, except that, generally speaking, they are not so advanced, and are of a more chronic nature.

Pachymeningitis was rarely met with in these cases; the brain was seldom greatly wasted, and if it were so, it was generally one hemisphere which was markedly affected, due to acute vascular changes affecting extensively one hemisphere, associated during life with epileptiform seizures and hemiparesis, aphasia, and, occasionally, hemianæsthesia and hemianopsy. The pia-arachnoid membranes were thickened in proportion to the atrophy in the usual situation—the fronto-central regions, but the thickening was seldom very marked. In some cases the naked-eye signs were so slight as to require microscopical examination for confirmation, and in these the clinical symptoms were usually hallucinations and delusions with little dementia. The dementia, as pointed out by Dr. Bolton, was found usually proportional to the degree of wasting, but not always. The most marked changes in these early cases was found both macroscopically and microscopically in the orbital and pre-frontal regions, which occasionally exhibited a granular surface like a cirrhotic kidney.

The lateral ventricles were dilated in proportion to the atrophy of the hemispheres. In several cases there was marked dilatation of the posterior part of the lateral ventricle, so that it extended within half an inch from the tip of the occipital lobe. This was found to be due to atrophy of the fronto-occipital and temporo-occipital fibres especially. The ependyma was usually granular, but not in the early cases;

the fourth ventricle exhibited dilatation of the lateral sacs, and granulation of the surface in every case. This condition, however, is met with, and, indeed, all the other naked-eye signs to some degree, in all cases of chronic insanity with wasting, but it is seldom that one finds granulation of the floor of the fourth ventricle covering the calamus scriptorius in any other affection than progressive paralysis; it was present in even the earliest cases of tabo-paralysis; when there was no appearance of granulation in the lateral ventricles it was found in this situation. In other brain diseases, in which there may be just as much wasting of nervous tissue, there is nothing like the same amount of ependymal change, consequently in this disease, progressive paralysis, there must be some difference in the cerebro-spinal fluid or the capacity of formative reaction of the lining epithelium and glia tissue to account for this excessive granulation. I regard this, like the filling up of the central canal of the spinal cord, as an exaggerated formative proliferation of embryonic and glia tissues brought about by irritation, and this fact is of importance when placed by the side of other facts denoting bio-chemical toxic irritation.

Vessels.—As I have shown elsewhere, if all the vessels of the brain, even in young people suffering with paralysis, be pulled out and washed with water, a few nodular patches of atheroma can be found in a considerable number of the cases, but this change may be regarded as merely a sign of previous syphilitic infection, and plays no part in the pathology of the morbid process. Only in cases of people who have passed 45 does one find any really marked change in the cerebral vessels, and then it is doubtful whether this change plays any important part in the production of the morbid process; it is merely a sign of pre-senile decay, and probably of the action of the syphilitic virus, especially when considered by the side of another fact, namely, the presence of atheroma of the aorta—a pathological condition found in the great majority of cases of progressive paralysis —more than 80 per cent. This condition is found even in young juvenile cases.

In none of the twenty-eight cases referred to in the

synopsis was there noted thrombosis of the veins opening into the longitudinal sinus, causing red softening; this, however, I have seen in cases of progressive paralysis giving rise to epileptiform seizures, unilateral or bilateral, according as the process affected one or both hemispheres, and followed by hemiplegia, first of one side, then of the other, causing death, usually with hyperpyrexia. Syphilitic endarteritis was not met with in any of these cases, but I have seen it several times in cases of progressive paralysis.

Microscopic characters of the disease may be considered under three headings:—(1) Changes in the nervous elements, cells, and fibres; (2) changes in the glia tissue; (3) changes in the vessels.

(1) I do not propose to give a long description of the changes met with in the nerve-cell, for they in no way differ from those met with in ordinary paralysis, and these changes are admirably described by Dr. Watson.

Comparing, however, a large number of sections of experimental anæmia, tabo-paralysis, and general paralysis, I have formed the conclusion that the changes in the nerve-cells are of two kinds, acute and chronic; that the chronic process begins in the superficial layers of the cortex, small and medium-sized pyramids, and the molecular layer. The changes are slow and insidious; the cells and their fibres undergo an atrophic process without any marked inflammatory reaction. The acute process, on the other hand, occurs in paroxysms, and is associated with the formation of an acute irritant toxin having a local action, and causing, partly by its own chemical properties and partly by the inflammatory reaction of the vessels with capillary obstruction, an acute coagulation necrosis of the cells very similar to that produced by ligature of the four arteries supplying the brain in cats and monkeys, which is invariably followed within twenty-four hours by a fatal result, preceded by epileptiform fits. (Dr. Leonard Hill's experiments.)

The cells stain diffusely, the Nissl granules are absent, and the chromophilous substance takes the form of a fine dust throughout the cytoplasm, the achromatic substance not being visible as the whole of the protoplasm is stained.

If Unna's polychrome or a mixture of methylene blue and saffranine be used, the cells are stained a pinkish purple, indicating an acidophil, instead of basophil reaction.

The nucleus is often swollen up and clear, the nucleolus staining deeply by contrast.

I have observed that the small and medium-sized pyramids, in cases with acute symptoms terminating fatally, show a very similar condition to that which I have observed in acute fatal experimental anæmia—namely, the nucleus swollen like a bladder to five or six times its size, clear, and surrounded by a thin film of cytoplasm ; the nucleolus deeply stained, the intranuclear network faintly. In proportion to the destruction of the nerve-cells, there is, as a rule, but not necessarily, an overgrowth of glia-cells and vascular changes, which will be shortly considered.

The neurons may be entirely destroyed, or only a small, irregular, triangular mass of protoplasm be left; or the larger processes may still persist, but present no Nissl granules, and exhibit no dendritic processes. Often the apical process is like a corkscrew; this condition I have found in imbecile brains, and I look upon it as evidence of a chronic process of atrophy.

Such morbid conditions destroy the normal lamination of the cortex and the characteristic columns of Meynert.

Occasionally I have observed cells with a nucleus undergoing division; this I have seen in absinthe poisoning, and I regard it as a sign of intense irritation. Salaman, in the first volume of the ARCHIVES, described a case of taboparalysis, in which he observed division of the nucleus in the cells of the posterior spinal ganglion.

As Tuczek pointed out, atrophy of the tangential and supraradial fibres is the earliest and most constant microscopic change met with in general paralysis. None of the twenty cases which were examined failed to show this change to some degree ; but I do not remember one case in which the Marchi method showed recent degeneration of the tangential system. Possibly the method does not lend itself to showing destruction of these very fine fibres—more likely, however, it is that the process has taken place, and the products of

destruction have been absorbed, or that as in tabes, the process is slow and progressive. When atrophic changes in these fibres could not be discovered in other regions of the brain, they were invariably found in the prefrontal, and usually in Broca's convolution. Many of these cases showed a marked atrophy of the tangential system in the ascending parietal and the paracentral lobule, more marked than in the ascending frontal. I thought possibly this might be associated with the fact of the degeneration of the afferent system, but Campbell has shown that the ascending frontal normally possesses a greater wealth of tangential fibres than the ascending parietal; it is likely that this is associated with the presence of the Betz cells, the apical processes of which terminate in a panniculus, as shown by Cajal, in the molecular layer. It was frequently noticed that in one section there would be a considerable local disappearance of tangential and supraradial fibres, and we must account for this by the fact that there was a local atrophy of cells corresponding thereto. In fact, Nissl preparations clearly show this, and frequently there is not a uniform disappearance of cells in the superficial layers, but frequently little foci of atrophy.

In Case 28, which we may consider as either one of tabes commencing with a mental crisis, or as tabo-paralysis with arrest of the cerebral disease and progress of the spinal, this unequal destruction of the tangential and supraradial fibres was especially observable. In that case, moreover, page 23, the atrophy and thickening of the membranes (see Plates I. and II.) were much more marked over the ascending parietal in its upper part than the corresponding portion of the ascending frontal. There were no acute changes in this case, and the superficial cells, beyond a certain amount of diminution in numbers and therefore thickness of lamination, presented no abnormal appearances. This was the only case observed which would at all correspond with cortical changes in tabes to which Jendrassik first called attention. Many people think that his cases were really tabo-paralytics, and I see no reason why they should not have been. For if Case 28 had died in the attack of mania, I should certainly have described it as a case of tabo-paralysis; instead of this, he survived thirteen years.

The atrophy of these superficial fibres is by no means limited to the fronto-central regions, although it is more marked there as a rule. Every part of the cerebral cortex may exhibit this change and sometimes it was found marked in the angular, temporal and occipital regions. In advanced cases, the interradial and, to a less degree, the radial fibres, were found atrophied, but as a rule these were not affected, excepting in cases of marked wasting of the brain. In staining by the Weigert method it was often found that the myelin did not take the stain readily, behaving in this respect like fœtal or infantile tissue and agreeing also with what was noted about the difficulty of staining the spinal cord in some of the cases. By the Marchi method the radial fibres were often found in a process of acute degeneration, especially where there was marked wasting of one or both hemispheres and signs of acute vascular changes. Such cases were found to show marked recent degeneration in the pyramidal systems in the cord, and indicated an acute death of the large Betz cells, from which these pyramidal fibres proceed. But we have seen that in twenty cases of the twenty-eight, there was degeneration and sclerosis of the crossed pyramidal tracts in the cord which could not be followed higher than the lower or mid-dorsal region. We can only account for this by a nutritive failure of the psycho-motor neurons, a chronic atrophic process affecting the most outlying portions of the neuron, most remote from the central seat of trophic influence, the nucleus of the cell. This presumably slow degenerative process of the crossed pyramidal tracts cannot be explained by any vascular or local condition of the spinal cord, or other long tracts in the lateral columns would be affected. The only explanation to my mind is the one I have given.

(2) *Glia proliferation.*—This is so very completely described by Dr. Watson that I need not do more than say my observations, made subsequent to his investigations, confirm the results which he has described. I hope to publish shortly also observations showing that atrophy produced by experimental lesions can produce active glia proliferation. There is one point, however, in connection

with glia formation which is not dealt with by Dr. Watson, and this relates to the evidence that the cerebro-spinal fluid in progresssive paralysis may contain some substances which, by irritation, produce formative proliferation. In three cases there was heterotopia spinalis; in one, Case 46, a careful study was made of the changes in the epithelium lining the dilated canal. By the Marchi method the epithelium was seen to contain numbers of black particles, presumably products of fatty degeneration. By Mallory stain a formative proliferation was apparent in this epithelium, not limited to the posterior surface where the tabic process in the posterior columns was adjoining, but affecting the whole epithelium. I mention this latter fact because it supports the view that this active formative proliferation was not related to a substitution process, but rather to an irritant action of the fluid contained in the central canal. It will be remembered that this case died in epileptiform seizures, and, as I will shortly point out, an acute vascular irritant formative process is the most significant of all the signs of progressive paralysis. Examination of this epithelium (see photomicrograph, Plate VIII., fig. 1) shows the nuclei of the cells undergoing an active proliferation; the nuclei, on account of the chromatin they contain, were deeply stained blue, and surrounded by a granulous pink-staining protoplasm, resembling the spongio-blasts of the embryonic tissue of the central nervous system.

The nuclei of these spongio-blasts can be seen dividing to form two or even a column of cells. From the base of the epithelial cells long fibrous strands, passing deep into the substance of the cord, can be seen, and in the spaces intervening these embryonic glia cells are packed. I could then trace, proceeding outwards from the central canal, successive changes in the appearance of the glia cells very similar to those described by Dr. Watson.

It is a well-known fact that in most chronic nervous diseases, and in old people, the central canal becomes filled up, but in quite young people suffering with general paralysis, or with Congo sickness, diseases in which there is other evidence of intense irritation, the central canal becomes

filled up, and its place marked by a great number of embryonic glia cells, similar to those which I have described above. The epithelium lining the canal, from which presumably these cells originated, has disappeared, owing to, probably, the pressure and consequent atrophy caused by proliferated glia cells.

(3) *Vessels.*—The most constant and striking change which can be observed in progressive paralysis is afforded by the appearances presented by the vessels. Under a low power in an acute severe case of this disease there is such a proliferation of cells on the vessel walls and in their surrounding lymphatics, which stain deeply with basophil dyes, that it almost appears as if the stained substance which was previously contained in the neurons had been transferred to these cells around the vessels. The more acute the symptoms and the more rapid the progress of the case, the more obvious is this vascular change. What is it? What does it signify?

Vogt has stated that plasma cells, which he regards as altered lymphocytes, are the cause of such an appearance of the vessels, and pathognomonic of the disease.

Mahaim, on the other hand, considers lymphocytes pathognomonic of the disease; Havet, although he admits that plasma cells are found in general paralysis in nearly all cases, does not admit that they are pathognomonic.

These plasma cells are not peculiar to the brain; they were first described by Unna, and he considered them as fixed tissue cells which have undergone proliferation. Marscholko has shown that they can be found in glanders, syphilitic granulomata, tubercle, lepra, carcinoma, and other diseases; and he believes they are altered lymphocytes.

The experiments of Cornil and Ranvier in the recently published edition of their "Pathology," show that irritation of the serous membranes by chemical substances can produce a proliferation of the endothelial cells in all respects resembling these plasma cells.

Pappenheim has recently introduced a differential stain whereby the nuclear chromatin substance is stained green and the surrounding granoplasm of the cell pink.

The ordinary Nissl method for staining nervous tissues was used by Vogt, thionin by Havet. Unna's polychrome is useful also, for it stains the granoplasm pink, the chromatin substance purple.

The Unna-Pappenheim stain, when it succeeds, shows more clearly than any other the morphological characters of the plasma cells; also their close relation to lymphocytes.

Lymphocytes.—The nuclear network of the lymphocyte is stained green; from a central nucleolus, like the spokes of a wheel, fine strands of chromatin extend to the nuclear membrane, ending generally in little knots. The nucleus, as a rule, is surrounded by hardly any cell protoplasm. Lymphocytes are not often seen in the blood contained in the vessels, but single, in pairs, or groups, in the perineuronal and perivascular spaces, in the cerebro-spinal fluid and subarachnoid space. They occur in normal brain tissue. Occasionally lymphocytes may be seen with a certain amount of granoplasm stained pink, partially surrounding, like a half-moon, the nucleus. These may be transitional, and probably represent young forms of the plasma cells of Marscholko.

Origin of the lymphocytes.—(*a*) The Blood. Dr. Pugh has found an increase of lymphocytes in the blood of epileptics during the fits. Dr. Boddington has kindly allowed me to refer to some unpublished observations which he has made, showing that during the second stage of general paralysis there is a considerable increase of the lymphocytes in the blood.

DR. BODDINGTON'S RESULTS.

[It is not certain that the increase per cubic mm. is due to an arterial increase, as the total volume of blood has not been measured; but it is remarkable that in these conditions it appears viscid, and flows with difficulty.]

This high percentage of polymorphonuclear cells during the seizures is a marked contrast to the condition found in cases of status epilepticus, in which, during the fits, there is an increase of mononuclear cells, chiefly lymphocytes, which latter may form 40 to 50 per cent. of the total.

Immediately following the status there is an increase of polymorphonuclear cells, even up to 90 per cent.; this, however, is quite transitory, lasting for twelve to twenty-four hours, whereas this high percentage in paralytic seizures continues for fourteen days, or more, and the return to the condition normal to the patient before his attack may take four or five weeks to

accomplish. Myelocytes to the amount of 1 per cent. are seen in status epilepticus.

In the second stage of general paralysis the blood differs from that of a healthy individual in that the percentage of mononuclear cells is increased so as to form 40 per cent. or more (in some few cases as much as 60 per cent.) of the total white cells, and of these considerably more than half are lymphocytes.

In the third stage there is an increase of polymorphonuclear cells,* which amount to 80 or 85 per cent. of the total white cells.

In the convulsive seizures of the second stage there is a very marked and sudden increase in the total number of cells per cubic mm., so as to form in the course of a few hours six or seven times the normal number; and this increase consists almost wholly of polymorphonuclear cells, which form 90 to 95 per cent. of the total. At the same time there is a total disappearance of eosinophils, and myelocytes appear in the blood.

Examination of sections of the brain from a large number of cases of various diseases in which there have been fits prior to death, including five cases of status epilepticus, has shown the existence of excess of lymphocytes in the perineuronal spaces. These may come from the blood, but lymphocytes are not usually regarded as capable of active migration through the vessel wall. They may, however, be derived from lymphocytes normally existing in the lymph spaces of the tissues. Pappenheim regards this as probable. We may therefore conclude that the altered condition of the blood which is associated with the fits may cause a stimulant and proliferative action on the lymphocytes of the blood, and especially at the primary seat of the morbid process—the brain.

After examination (by one of the methods related) of an immense number of sections of the brain and spinal cord of the various forms of nervous disease and experimental lesions enumerated below :—

Twenty cases of experimental anæmia (dogs, cats and monkeys), by Dr. Leonard Hill.

Cases of abrin, ricin and botulin poisoning, by Dr. Durham.

One case of sunstroke.

Two of pernicious anæmia.

One stab in the heart (probably man suffering with acute alcoholism).

One leukæmia.

One exophthalmic goitre.

* The polymorphonuclear cells may, however, be due to terminal infection. (F. W. M.)

Two beri-beri.
Five status epilepticus.
Two acute alcoholism.
Five alcoholic dementia and polyneuritis.
Two amyotrophic lateral sclerosis.
Five cases of juvenile paralysis.
Twenty cases of tabo-paralysis.
Ten cases of general paralysis.
Two cases of Congo sickness.
Two cases of multiple syphilitic gummata.

I came to the conclusion that the plasma cells of Marscholko described by Vogt as pathognomonic of general paralysis, and which will now be described, are almost pathognomonic of this disease. I say almost, because I found them in a case of Congo sicknesss with fits (*vide* fig. 7, Plate XI.), and I failed to find them in one case of juvenile general paralysis. These cells contain a nucleus very like the nucleus of a fully-developed lymphocyte, viz., a central nucleolus with wheel-like strands of chromatin ending in four to seven knobs beneath the nuclear membrane. Like the nucleus of the lymphocyte, the chromatin stains deeply green, whereas the granoplasm around the nucleus is stained pink by Pappenheim's method: very frequently there is a clear halo between the nucleus and the granoplasm. The plasma cells are of varying size, from 7 to 14 μ, they are polygonal, oblong, oval or triangular in shape; the green-stained nucleus is usually at one end of the cell; frequently two cells having their nuclei in opposition indicate cleavage. Many of the cells show an endogenous nuclear proliferation. The granoplasm contains a fine dust of particles stained pink and no coarse granules, so that it has a grumulous appearance. These cells lie like a plaster on the vessel wall (*vide* figs. 3, 5, 6, 7, Plate VIII.).

In the neighbourhood of vessels so affected one always find a proportionally marked acute destruction change in the neurons.

In some cases of tabo-paralysis in which there had been no fits, and death had occurred within a short time of the onset of mental symptoms, the prefrontal and orbital regions of

the cortex alone may show naked-eye changes of the lesions characteristic of general paralysis, and it may *be only in such regions that the plasma cells will be found.* (Case 39, p. 156.)

In cases where there has been marked spinal affection these cells have been found in great abundance around the vessels in Broca's convolution, the island of Reil and the posterior third of the first temporal. (Cases 46 and 54.)

We may associate their existence in abundance with intense irritation, increased neuronic irritability followed by degenerative destruction. The former case was not paretic in the limbs; his speech, however, was markedly affected, and facial paresis existed. Accordingly, marked plasma cell proliferation and acute neuronic destruction was found in Broca's convolution and the lower part of the frontal and parietal convolutions, and but little in the second frontal and adjacent motor area. In Case 54, on the other hand, there was marked sensory aphasia, hemiparesis, and transitory hemianæsthesia ; plasma cells in abundance and acute cell destruction were found in the angular, supramarginal and first temporal of the left hemisphere.

Plasma Cells proportional to Acute Neuron Irritation and Destruction.

The plasma cells of Marscholko were found in all cases of general paralysis and tabo-paralysis examined; they were not found in the brain of any other of the conditions enumerated, with the exception of one case of Congo sickness, and one of multiple syphilitic gummata. They were not numerous in the Congo sickness, and required much search (*vide* fig. 7, Plate VIII.), although the perivascular lymphatics throughout the whole central nervous system were crowded with lymphocytes in a way that I have never seen in any other disease. Sections of the spinal cord, medulla, and pons in tabo-paralysis and general paralysis did not show these plasma cells around the vessels.

They exist especially in those regions (fronto-central) of the brain which generally show wasting in general paralysis, and their abundance is clearly associated with the amount

and the intensity of the acute neuronic irritation and destruction.

Endothelial Cells as the Origin of the Plasma Cells.

The endothelial cell proliferation is marked in all cases where there is active destruction going on. The nuclei of the capillaries increase in numbers and can be seen undergoing mitosis. This proliferation of the endothelial cells would tend to obliterate the lumen of these delicate tubules and obstruct the passage of red corpuscles, thus interfering with tissue respiration and the removal of the waste products of neuronic activity. Endothelial cells proliferate in the vessels of the spinal cord, or at any rate, there is an increase of the nuclei; but I have never observed plasma cells in the gray matter of the spinal cords of paralysis cases. A good way to study the endothelial proliferation of the capillaries is to make film preparations of fresh cortex squeezed between two cover-glasses (*vide* fig. 4, Plate VIII.). The capillaries can be traced into the venules; the proliferated endothelial cells can thus be determined. The walls are no longer clear with a delicate outline, but consist of a granoplasm with increased numbers of long nuclei. As you pass from the capillary to the venule the nuclei become more oval, and the proliferated cells take on the form of plasma cells: but it may be argued that these cells may arise from the endothelial cells of the lymphatics surrounding the small venules, or lymphocytes contained in the sheath.

I have observed plasma cells in the perineuronic spaces and in the tissue away from vessels; moreover, the much closer resemblance of the nucleus of the lymphocyte in shape and arrangement of the chromatin, together with the occurrence of transitional forms between lymphocytes and plasma cells, inclines me to alter my original opinion and conclude that the balance of evidence is in favour of the plasma cells being derived from lymphocytes and not from endothelial cells, although the latter undoubtedly undergo active proliferation.

The proliferated endothelial and plasma cells seem to

have a phagocytic function, for we can find blood pigment in various stages of disintegration and chemical change within them.

What is the pathological significance of these plasma cells in the brain in general paralysis?

The clinical symptoms associated with the anatomical findings undoubtedly show an intense irritant morbid process, followed by acute neuronic destruction. But, as already remarked, plasma cells are found in many other morbid processes where acute irritation and destruction is occurring; therefore in general paralysis they are only indicative of an acute irritative process in the brain.

We have next to consider the source of the irritant, which, presumably, is of a bio-chemical nature, and produced paroxysmally by *a localised conspiracy of factors*; for if some toxin or autotoxin generalised in the blood were the sole factor, then we ought to find its effects manifested equally throughout the nervous system, but it is not.

Those portions of the hemispheres, the veins of which drain into the longitudinal sinus, are the regions in which venous stasis is most apt to occur, for the following reasons: The blood flows against gravity; the veins embouch into the longitudinal sinus in a contrary direction to the current; the suction action of the thorax influences the flow from the lateral sinuses first; consequently violent expiratory efforts and suspension of respiration leading to cerebral congestion, or any general condition of venous plethora from portal congestion, would, owing to these mechanical disadvantages, tend to stasis in the fronto-central regions. Again, the brain, being contained in a closed cavity, the quantity of blood in the organ is always the same, therefore if vaso-motor irritation causes sudden anæmia from diminution of arterial supply, a proportional reflux of venous blood would take place and tend to flow into these regions. Now the portion of the brain most likely to suffer from sudden anæmia is the portion supplied by the internal carotid (viz., fronto-central), because the force of gravity would operate most on the blood contained in the distribution of this vessel. Therefore the morphological conditions of the arterial and

venous circulation in the fronto-central regions favour anæmia and venous congestion.

Slight faints and lapses of consciousness are among the earliest prodromal signs of general paralysis, and are indicative of these vascular disturbances. These, however, are subsidiary, but important exciting and determining factors in the disease.

The paroxysmal character of seizures, followed by apparent betterment, the microscopical evidence of an intense irritative and destructive process more obvious than in almost any other brain disease, *point to the formation in the blood of a toxin which fixes on to certain portions of the central nervous system*, which either stress, heredity, or anatomical conditions place in a lowered state of resistance. If the vascular factor alone were a cause, heart disease should be a cause of general paralysis. The paroxysmal attacks in tabes, of lightning pains, of visceral crises, of mental crises, followed by intervals of relief, all point to some coming and going of an irritant which acts upon decaying structures.

The mania and grandiose delusions which frequently subside, and the paroxysmal character of the epileptiform seizures, point to the formation of a toxin or accumulation of a toxin in the blood, which produces an effect, but is destroyed to accumulate again. The observations of Krafft-Ebing strongly support the view that general paralytics possess an immunity to syphilis; it may be presumed that the effects of the toxin are still in operation. He inoculated eight general paralytics, who exhibited no signs of syphilis, with the virus of a hard chancre, and they showed no signs of infection subsequently when watched for 180 days.

As in diphtheria, the general toxic action of syphilis altogether overshadows, and, indeed, often bears no proportional relationship to the local effect of the contagium, especially in the late after-effects. Experiments of Ehrlich, confirmed by Bulloch, seem to show that there are a mixture of bodies in the crude toxins of diphtheria. Likewise Ehrlich and Madsen have shown that certain bouillon cultures of the tetanus bacillus may contain two toxins, a

tetano-lysin, and the ordinary spasm producing body tetano-spasmin. Unfortunately, owing to the absence of our knowledge of the specific germ of syphilis and the immunity of all animals to infection, it can only be deduced by analogy that Hitzig is probably right in supposing that the syphilitic virus may convey several poisons, one of which may be latent, and produce these late manifestations, tabes and general paralysis. Like diphtheritic paralysis, these late nervous manifestations bear no necessary relation to the primary local and general constitutional manifestations accompanying the disease. The only difference in the analogy between syphilis and diphtheria is the much slower evolution of the symptoms of the former, and it may be added, as a result, the life-long immunity conferred thereby.

Is it therefore warrantable upon the facts to hypothecate a latent toxin of syphilis which has a special affinity for nervous structures—a toxin which can only operate under certain abnormal metabolic conditions of the neurons. In Professor Welch's most illuminating Huxley lecture he thus referred to the work of Ehrlich and Preston Kyes :—

"Preston Kyes, working in Professor Ehrlich's laboratory, in an investigation just published on the mode of action of cobra venom, confirms the conclusion of Flexner and Noguchi concerning the amboceptor nature of cobra venom. . . . Of great significance is the demonstration by Kyes of still a third substance, namely, lecithin, which is capable, through combination with the venom intermediary body, of completing the hæmolytic potency of venom. . . . The suggestion by Ehrlich and Kyes that possibly the cholin group is the toxophore group of lecithin is interesting. . . .

"Flexner and Noguchi have demonstrated experimentally that, like the hæmolytic, so also the leuco-toxic, neuro-toxic and other cyto-toxic properties depend upon combinations of venom intermediary bodies, with complements contained in the cells poisoned by venom, or in the fluids bathing these cells. The positive demonstration by Preston Kyes of a special class of intracellular complements or endo-complements is unquestionably of great pathological interest, and seems destined to play an important part in many morbid conditions, both with endogenic and exogenic intoxication."

The products of degeneration of nervous tissues are numerous, and consist not only of cholin, but a number of bodies of the lecithin group, being various derivations of protagon. Cholin is the most easily separated and recognised physiologically and chemically, and it is possible that the products of degeneration vary according to the cause and nature of the destructive process. Still there is no evidence to show that these products of degeneration can *per se* produce the clinical manifestations and morphological changes indicating neuronic irritation and destruction of general paralysis, otherwise we ought to get these changes in other diseases, also destructive lesions of the nervous system. Therefore I think it may be conceived as possible that there is a latent toxin in the blood which combines with endo-complements the products of deranged neuron activity, producing locally (that is, where the neuron metabolism is deranged either by stress, circulatory deficiencies, or hereditary physiological or anatomical defects) an active neurolysin proportional to (*a*) the amount of latent toxin in the blood, and (*b*) the amount of endo-complement produced by deranged neuron metabolism. The glia-cell, lymphocyte and endothelial cell proliferation and the presence of abundance of plasma cells are the signs of the local vital reactions of the tissues to this poison and proportional to its amount and intensity. Where nervous tissues are latest developed and most unstable, where venous stasis and anæmia are most likely to occur, and in those structures which are subjected to the greatest physiological activity, there is defective metabolism to be found, and there, on this hypothesis, is formed an active toxin-producing irritation and destructive local effects. The nature of the conspiracy of factors and the vital reaction of the tissues will determine the seat and extent of the acute neuronic irritation and destruction.

Case.	Spinal Symptoms.	Mental Symptoms especially Progressive Dementia.	Speech Affection Verbal and Written.	Optic Atrophy.	Naked-Eye Signs of G.P.	Evidence of G.P.			Direct Pyramidal Tracts.		Crossed Pyramidal Tracts.		Exogenous Fibres.						Fibres.	
						Nerve-Cell and Fibre Degeneration.	Vascular Changes.	Glia Proliferation.					Post. Int. Tracts.	Post. Ext. Tracts.	Post. Int. Zones.	Charcot's Root Zones.	Lissauer's Tract.	Posterior Roots.	Ascending.	Descending.
28 ♂ ..	V M	S	O	Blind	m	m	O	m	O	O	O	O	V M	V M	V M	V M	m	V M	S	M
31 ♀ ..	V M	M	S	m	M	M	M	M	O	O	S	S	V M	V M	V M	V M	V M	V M	S	m
33 ♂ ..	m	M	M	?	M	—	—	—	O	O	S	S	m	m	m	m	m	m	O	O
34 ♂ ..	M	V M	M	?	M	—	—	—	S	S	m	m	M	S	m	M	m	M	O	m
39 ♂ ..	V M	m	m	M	m	m	m	m	O	O	m	m	V M	M	M	V M	M	V M	S	m
40 ♂ ..	V M	S	O	?	m	S	—	S	O	O	S	M	V M	M	M	V M	M	V M	S	M
†41 ♂ ..	m	M	S	?	m	m	M	m	O	O	m	m	M	S	m	M	m	M	O	S ?
42 ♂ ..	m	M	—	V M	M	M	S	m	S	S	m	m	M	S	m	M	M	M	O	S
44 ♂ ..	m	M	V M	?	M	M	M	M	O	O	—	M	M	m	m	M	S	M	O	O
45 ♂ ..	M	M fits	S	M	V M	V M	V M	m	M	m	m	M	M	m	m	M	M	M	O	m
†46 ♂ ..	M	M	M	O	M	M	V M	M	O	O	O	O	V M	M	M	V M	M	V M	m	M
47 ♂ ..	M	M	—	?	m	m	m	m	O	O	O	O	V M	M	M	V M	M	V M	S	m
48 ♂ ..	M	m fits	m	M	M	M	M	M	O	O	m	m	V M	M	M	V M	M	V M	O	m
49 ♂ ..	V M	M	S	Blind	M	m	V M	m	S	S	S	S	V M	V M	V M	V M	M	V M	M	V M
51 ♂ ..	M	M	M	?	M	M	M	M	O	O	S	S	V M	M	m	V M	M	V M	O	m
52 ♂ ..	m	M	M	?	M	—	—	—	S	S	m	m	M	S	m	M	O	O	O	S
54 ♂ ..	m	M	V M	? D A	M	V M	V M	M	V M	m	m	V M	M	M	m	M	m	M	O	O
55 ♂ ..	M	M	M	?	M	—	—	—	O	—	—	—	M	m	m	M	m	M	O	S
57 ♂ ..	M	M	S	?	M	M	V M	M	O	O	m	m	M	m	m	M	m	M	O	S
59 ♂ ..	m	m	m	Blind	M	m	m	m	O	O	S	S	M	S	S	M	S	m	O	O
†61 ♂ ..	m	M	M	V M	M	M	M	M	O	O	M	M	m	S	O	M	S	m	O	O
62 ♂ ..	M	M	S	Blind & deaf	m	m	m	m	O	O	O	O	V M	V M	M	V M	m	V M	S	m
63 ♂ ..	S	M	M	Blind	V M	M	M	M	O	O	S	S	m	S	S	m	S	m	O	O
64 ♀ ..	m	M	m	Blind	M	—	—	—	O	O	S	S	M	m	S	M	m	M	O	O
65 ♂ ..	m	M	m	Blind	M	m	S	m	S	O	O	M	M	S	S	M	m	M	O	S
66 ♀ ..	m	M	m	?	m	—	—	—	S	S	m	m	M	—	—	—	—	—	—	—
68 ♂ ♀	m	m	S	Blind optic-atro'y	m	—	—	—	S	S	m	m	M	m	S	M	S	M	—	—
Not recorded ♀	M	M	M	?	M	M	m	m	S	S	m	m	V M	V M	V M	V M	M	V M	M	V M

O = nil. D A = Acute Degeneration. S = Slight Degeneration. m = Moderate. M = Marked. V M = Very marked.
† Heterotopia spinalis. ? = Optic nerves not examined microscopically, but no naked-eye change noted. — = Uncertain or not ascertained.

This Table is only approximately true, because different levels of the cord may be affected differently in different cases.

Plate VIII

Fig. 1.—Photomicrograph of a section of the central canal of the spinal cord with heterotopia, Case 46. The nuclei of the epithelial cells, with their chromatin network, can be seen proliferating; lower down they can be seen surrounded by protoplasm, and undergoing active division. A column of four nuclei surrounded by protoplasm is seen; the upper two nuclei show very clearly two asters, indicating the nuclear division which has occurred. Mallory stain. Magnification, 750 diameters.

Fig. 2.—Photomicrograph of a section of the ascending parietal convolution from Case 45, showing a small vein surrounded by plasma cells, which are lying in a dilated lymphatic. The vessel at another part had ruptured and filled the lymphatic sheath with blood corpuscles. Some of the large, swollen-up cells showed in their interior the blood pigment in various stages of destructive disintegration; they appear to have, therefore, a phagocytic function. Nissl stain. Magnification, 500 diameters.

Fig. 3.—Photomicrograph of a section of Broca's convolution, Case 46, showing abundance of typical plasma cells of Marschalko. The cells are oblong, oval, polygonal, and triangular. The granoplasm is dark on account of its taking the pink dye. The nucleus is distinct, with its nucleolus and chromatin strands terminating in chromatin knobs. The nucleus is generally at one end of the cell, indicating active division. In some there is a clear halo around the nucleus. Many of the cells show endogenous nuclear proliferation. The cells are seen stuck on the vessel wall like the corrugated bark of a tree. At the lower end of the section the indistinct forms of the unstained red corpuscles are seen within the lumen of the cut vessel. Polychrome Nissl stain. Magnification, 750 diameters.

Fig. 4.—Photomicrograph of a film preparation of the vessels stained by Nissl method from a case of general paralysis. It shows the proliferation of the nuclei of the capillaries. the absence of red corpuscles, and the obliteration of the lumen by the endothelial cell proliferation. Magnification, 500 diameters.

Figs. 5 and 6.—Drawings to show different types of cells around vessels in general paralysis; p = plasma cell, m = mastzell, l = lymphocyte. Magnification, 600 diameters.

Fig. 7.—Photomicrograph of small vein, case of Congo sickness, stained by Nissl method, showing plasma cells. On account of the specimen being somewhat faded, the negative was not strong; the cells have been retouched, but it represents fairly accurately what could be seen. Magnification, 750 diameters.

PLATE VIII.

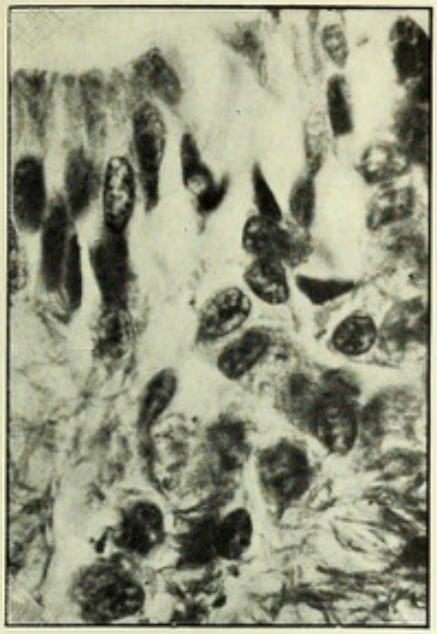

FIG. 1.

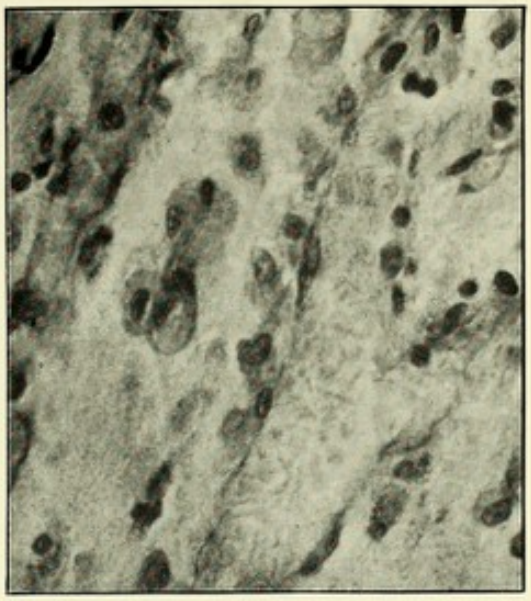

FIG. 2.

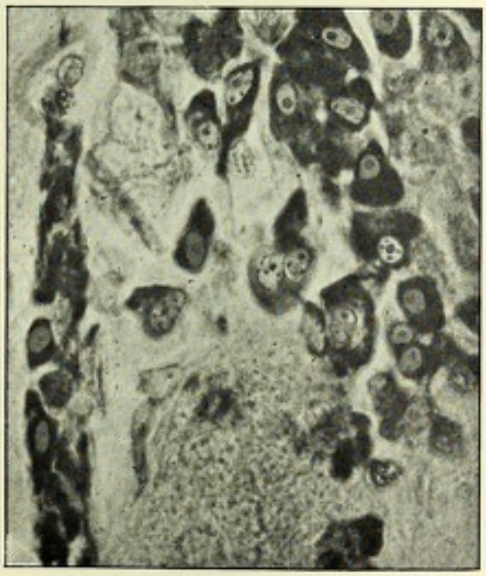

FIG. 3.

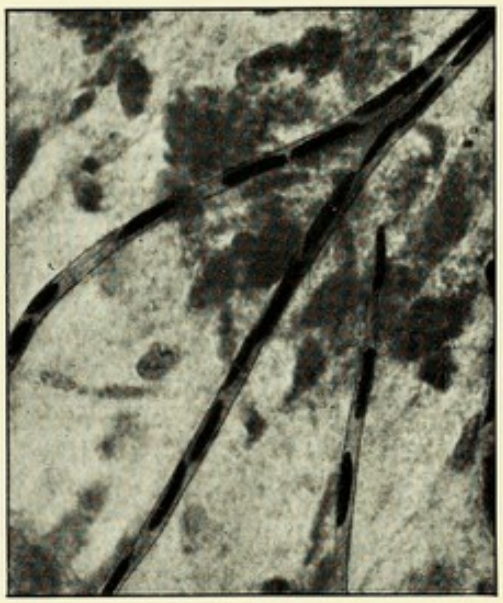

FIG. 4.

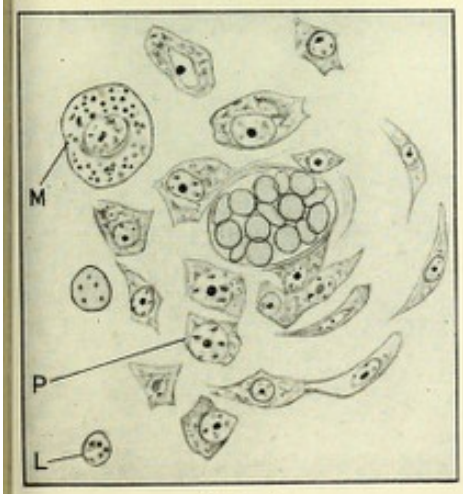

FIG. 5.

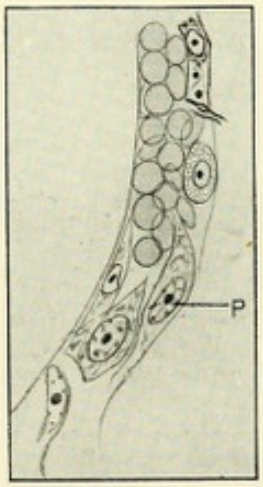

FIG. 6.

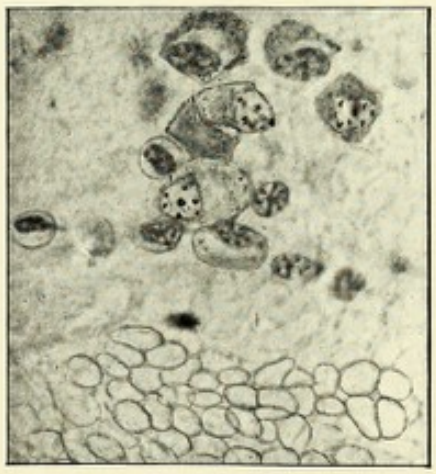

FIG. 7.

TABES IN ASYLUM AND HOSPITAL PRACTICE.

To face p. 306.

Notes upon the Pathology of Tabes and Tabo-Paralysis.

Leyden was the first to point out that the pathology of tabes depended upon the degeneration of the posterior roots and their intraspinal connections. Westphal, Dejerine and Oppenheim, among others, showed that the peripheral nerves were also affected; but most observers now agree with Leyden that the symptoms of tabes are especially due to the degeneration of the central projections of the posterior spinal neurons.

Every posterior root furnishes fibres to different sensory nerves of the periphery. The cutaneous surface is subdivided into zones or segments, of which each receives its sensory innervation from certain definite posterior roots. These segmental posterior root areas overlap one another, and each receives fibres from two or three contiguous roots. It will thus be manifest that the area of distribution of cutaneous anæsthesia will be quite different, when it depends upon alteration of the posterior roots and their intramedullary prolongations, to when it depends upon alteration of the peripheral nerves. In tabes, cutaneous sensory dissociation, especially in not very advanced cases, is the rule; whereas it is the exception for an anæsthesia of peripheral origin to exhibit the characters of dissociation. There is, however, not only dissociation of cutaneous sensibility, but also marked subjective and objective sensory disturbances of the deep structures, which may exist independently of cutaneous disturbances. We have already referred to the fact that probably the fine fibres of the posterior roots convey cutaneous sensations, and the coarse fibres sensations from the deep structures; and we have seen, moreover, that clinical observations and morbid anatomy have demonstrated that these two systems may be affected independently.

The interesting observations of Dr. Anderson upon the myelination of the different systems of somatic and splanchnic fibres are of great interest, and should help to throw light on some of the phenomena of this disease. He has shown that both somatic and splanchnic myelinated afferent fibres consist of two distinct sets, which develop

their myelin sheath simultaneously in both splanchnic and somatic posterior roots and peripheral nerves; the two sets of fibres convey impulses respectively from deep structures and cutaneous or epithelial structures.

Taking the myelinated splanchnic fibres, he finds that there is one set which has as its peripheral termination Pacinian corpuscles, contained in the structures of the abdominal viscera and larynx. These *large fibres* acquire their medullary sheath contemporaneously with the large myelinated fibres, terminating in the Pacinian corpuscles, muscle spindles and end-organs, which are contained in the deep somatic structures. The finer medullated fibres which terminate in the skin, and which are connected with pressure and painful sensations especially, are myelinated contemporaneously with the *fine set* of medullated splanchnic afferent fibres, which, he argues, supply the epithelial structures of the viscera, and which would endow them with common sensibility. Anderson considers that there is a correlation between the function of the afferent splanchnic and somatic fibres, which have Pacinian corpuscles as end-organs. The irritation of these neurons, he considers, gives rise to the deep pains in the limbs and the pains of the visceral crises.

Microscopic examination of the white rami shows that there are both large and small white medullated fibres contained in them; the large fibres correspond to these large splanchnic afferent fibres from the Pacinian corpuscles. The small fibres are partly efferent, arising from cells of the lateral horn of the intermedio lateral tract, which cells have their long axis directed horizontally, and are found in the thoracic region only, where this outflow of fine efferent medullated fibres takes place. Some of the fine fibres, however, are sensory fibres of the viscera, and subserve a protective function by virtue of the pain which intense stimulation, mechanical or chemical, might cause.

Normally we feel no sensation from our internal organs; when painful stimuli are conveyed by the white rami to the posterior spinal ganglia, thence to the spinal cord, where these flow over into the terminals of the somatic skin

neurons, they give rise to characteristic pains referred to morphologically correlated skin areas (Mackenzie, Head). Thus we may really have an illusion as to the true seat of the pain and the nature of the process occasioning it; our experience, however, will lead to its correction.

Besides pain, there is rigidity of correlated muscles due to a protective reflex, by which the parts are kept as much at rest as possible. The severe pain accompanying it may, as Prof. Sherrington aptly says, be looked upon "as a psychical adjunct of this protective reflex." The pain is, however, much greater in some individuals than others; not because the stimulus is greater, but because the subjective mental attitude of the individual, by a concentration of consciousness to the painful spot, is greater. But it might logically be asked, would it be possible to concentrate the attention on a viscus of which one has no knowledge by touch or sight, if it were not for the pain referred to the correlated skin area? By no effort of the attention can we imagine the rythmical contractions which are taking place with phasic or periodic regularity in our ureters, bile ducts and gall bladders. It is only when they contract violently in response to intense irritation, that we are painfully instructed by experience and the referred pain, as to the cause.

A great deal of discussion has arisen as to whether the viscera are really painful. I think there can be no doubt that a peculiar dragging and sinking sensation is associated with irritation of the viscera, probably due to stimulation of coarse afferent fibres with Pacinian corpuscles, independent of the sensation in the skin areas. Many of the patients who suffered with gastric crises complained of a deep-seated pain within the abdomen, and no doubt this was due to irritation of the splanchnic afferent neurons which were undergoing destruction in the posterior roots of the mid-thoracic segments.

The accompanying figure (47) illustrates this point.

Cases often attend hospitals for gastric troubles which are thought to be dyspepsia, whereas really they are very mild gastric crises. In severe cases there is no doubt intense

irritation, generally with destruction of the sixth, seventh, and eighth posterior roots, conveying afferent splanchnic

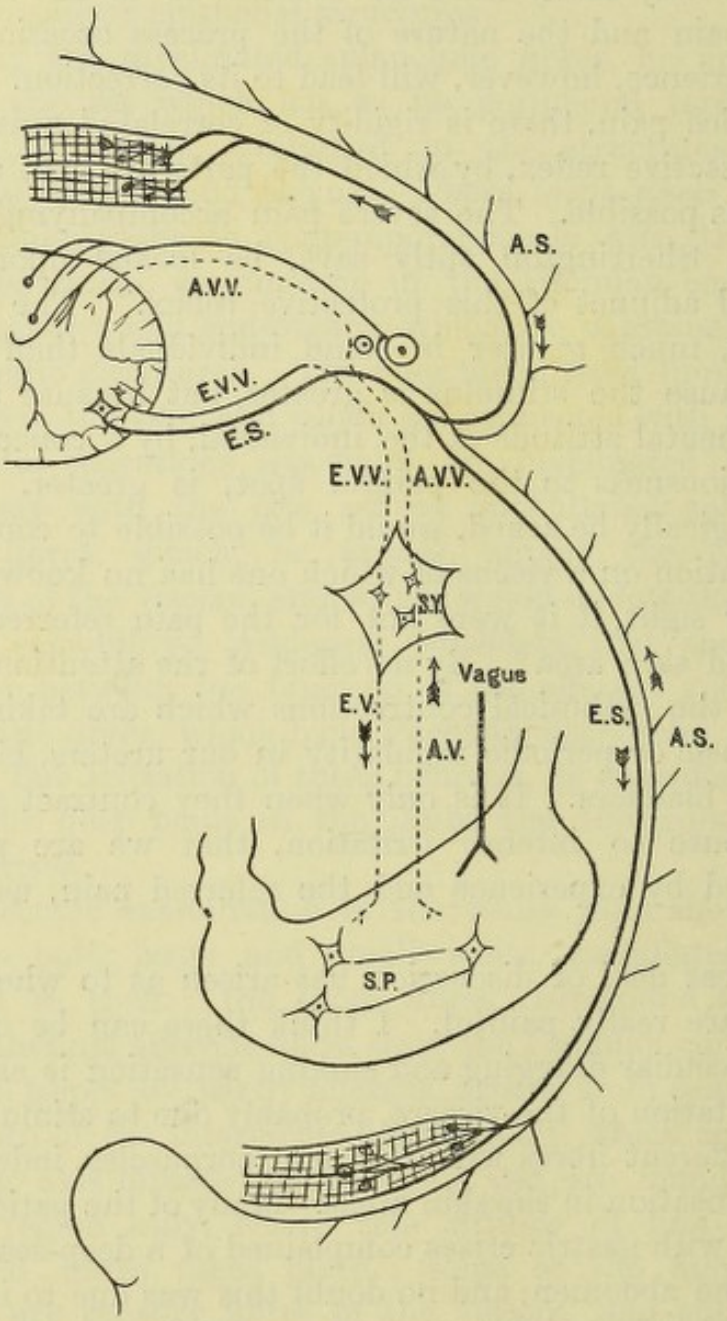

Fig. 47.

Diagram to show somatic and splanchnic nervous structures in the seventh thoracic metamere. The correlation of the skin and muscle in the protective reflex is indicated.

E.V.V. = Efferent splanchnic ; E.S. = Efferent somatic ; A.V.V. = Afferent splanchnic ; A.S. = Afferent somatic ; S.P. = Plexus of Auerbach and Meissner ; E.V. = Efferent visceral from sympathetic ganglion ; A.V. = Afferent visceral to sympathetic ganglion.

The coarse and fine fibres which exist in both somatic and splanchnic nerves are not differentiated.

impressions from the stomach (Case 31). The quality of a sensation depends on multiple circumstances—the nature intensity of the peripheral excitation, the state of the conducting neurons and of the cortical perceptive centres; but whatever be the cause of the objective sensation coming from the periphery, it is in the cortical terminus of the afferent system that the stimulus is perceived as a state of sensation of heat, cold, pain, contact or movement, and localised to a particular part of the body. The localisation is very often imperfect; this is explicable by the fact that each spinal segment of the grey matter of the posterior horns is correlated with a definite skin area. When all the sensory cutaneous neurons proceeding directly to that area have been destroyed, sensory impulses may still enter the cord, owing to the overlapping of adjacent roots in the nervous supply of that skin area, and the stimulus be referred to an area above or below. In the limbs, where the segmental character is obscured, the cause of this imperfect localisation is not so obvious. The wrong localisation is, however, due to a state of consciousness brought about by excitation of neurons in the wrong segment, and consequently is outwardly projected in the mind to that area of skin which is morphologically correlated with the segment excited.

The subjective attitude of the individual (borrowing an expression of Mr. Shand) plays a very important *rôle*, not only in the subjective disturbances of tabes and taboparalysis, but also in the objective; for often the objective sensory disturbances are erratic and changeable. Thus, where at one moment a patch of anæsthesia or analgesia is found, a little later the same patch may be sensifacient, or even hyperæsthetic or hyperalgesic. This may partly be accounted for by summation of stimulus, or by the variability in the irritability and conductibility of the systems of spinal sensory neurons, but still more often to the subjective attitude of the individual—the state of consciousness which is especially related to the physiological activity of the cortex cerebri.

In tabes with insanity, we can therefore understand how acute the suffering of a patient is, when he concentrates the

consciousness of an unsound, but still active, mind upon the pains and tortures, which he believes enemies and unseen agencies are working upon him. The effect on the mind is reflected in the physiognomy, which portrays misery and dejection.

In tabo-paralysis, in the early stages, there may be an intensification of the pains and sufferings by the subjective attitude of the individual towards the effects produced by the irritation and degeneration of the sensory, somatic, and visceral neurons; and as we have said before, and given numerous illustrations thereof, such may cause pseudo-hallucinations, illusions and delusions.

In the later stages of tabo-paralysis, as, indeed, in general paralysis, when the dementia becomes pronounced, the patient suffers very little or no pains, nor does he feel pain when pricked with a needle. The stimulus may be the same, but the effect on consciousness is diminished or lost by the partial destruction of the cerebral cortex. This probably explains the fact that such patients may often suffer with very painful diseases without any complaint. The absence of pain associated with physical signs of tabes suggests, therefore, cortical degeneration.

Coordination.

Definition.—Coordination is the regulation and adjustment of the innervation currents flowing to correlated groups of muscles in such a way that the movement may be executed with precision in rate, force and direction, with the least expenditure of nervous and muscular energy. To attain perfect coordination even in the simplest movement entails a vast complexus of functionally correlated neurons in the cortex, mesencephalon, cerebellum and spinal cord.

The current of reciprocal innervation of correlated groups of muscles is dependent directly upon the outflow from correlated groups of spinal-motor neurons, with which the muscles are in direct anatomical connection.

The complexus of neurons which determines, controls, adjusts and regulates this mechanism of innervation is much more complicated than is generally supposed and taught.

Every day new experiments and observations show fresh paths indicating groups of neurons with special functions. We may consider these groups, however, under three headings:

(1) Spinal, afferent and efferent.
(2) Cerebellar and mesencephalic, afferent and efferent.
(3) Cerebral, afferent and efferent.

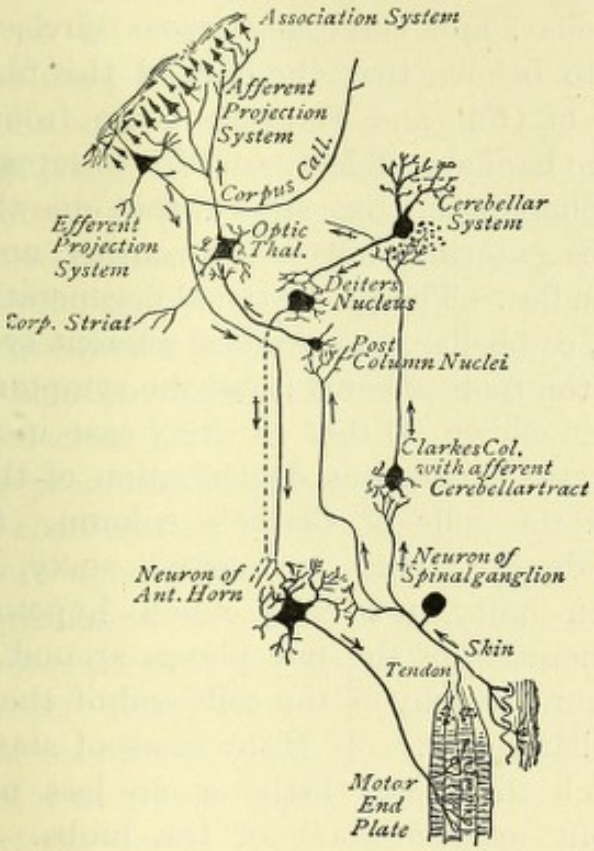

FIG. 48.

These are represented as the nervous circles in the accompanying diagram, which, however, is considerably simplified. In each of these nervous circles there are complex *association* systems, especially in the cerebral. The spinal intercalary are not represented, nor are the neurons which convey impulses by the optic nerves to the mesencephalon and cortex, nor those coming from the semicircular canals to Deiter's nucleus.

In tabes no two cases are exactly alike as regards the clinical phenomena and morbid anatomy, but there is a

general resemblance in all, for of the 28 cases I have examined, the three sets of coarse fibres entering the posterior columns invariably showed simultaneous degeneration.

Reasons have already been advanced to show that these three sets of fibres, proceeding respectively to the spinal motor cells, to the cells of Clarke's column, and to the posterior column nuclei, represent sensory channels in the spinal, cerebellar, and cerebral nervous circles. There is good reason to believe that the bulk of the fibres forming the columns of Goll and Burdach come from the small muscles of the hands and feet, and this is supported by the fact, which Sherrington has shown, that one-third to one-half the fibres entering muscle are sensory, and terminate in muscle spindles. The proportional degeneration of fibres in the spinal, cerebellar, and cerebral afferent systems varies according to the roots affected ; and the symptoms vary too.

It has been shown (1) that in every case in which there was marked ataxy there was degeneration of the plexus of fibres around the cells of Clarke's column. (2) That in Friedreich's disease there is marked ataxy, no loss of cutaneous sensibility, not very much hypotonus, but a marked degeneration of the fine plexus around the cells of Clarke's column, atrophy of the cells and of the ventral and dorsal cerebellar tracts. (3) Many cases of ataxy were met with in which there was little or no loss of cutaneous sensibility, but marked ataxy of the limbs. Again, the destruction of the coarse fibres in the lumbo-sacral region was usually much in excess of that of the fine fibre system of Lissauer's tract. We therefore conclude that the cerebellar afferent impulses play a most important part in co-ordination, and that the impressions coming from the deep structures are of more importance in coordination than those from the superficial structures.

We can now consider the mechanism by which the *neuron complex* represented in the diagram consciously and semi-automatically regulates and adjusts the innervation of muscles. We are conscious of the position of our limbs by the sensory impulses arriving at the seat of consciousness,

the cerebral cortex; and before a movement can be initiated ideation of the present position, followed by revival in consciousness of the position willed, precedes the outflow of impulses through the psycho-motor neurons. The accuracy of the kinæsthetic memory picture therefore determines the perfection of the adjustment and regulation of the efferent impulses concerned in the coordinate movement. The anatomical substratum of cerebral coordination is therefore formed of all those neurons which convey sensory impulses from the periphery to the cortex, of the cortical perceptive neurons, and of the infinite combinations of association neurons lying between them and the psychomotor neurons, all of which give rise to the memory pattern which plays upon the terminals of the dendrons of appropriate groups of psycho-motor neurons, the efferent stimuli from which cause reciprocal innervation of correlated groups of spinal motor neurons, which in their turn preside over groups of synergic muscles.

With the progressive evolution of the brain in the zoological scale, coordination in successive movements depends primarily more upon cortical and less upon spinal action. In man, coordination is primarily cerebral, even in semi-automatic processes such as walking; all spinal coordination has not, however, disappeared, for the most perfect example of subcortical coordinate reflex is still left to man in the prehension of a new-born infant, as shown by the fact that it can support the weight of its body when grasping a bar.

When the crura cerebri are divided in animals, viz., apes, decerebrate rigidity is produced; the cerebral nervous circle is no longer in operation, only the subcortical circles; and certain phenomena of great interest can be demonstrated. If the skin of the palm of the hand be stimulated by dipping in hot water, *a reflex coordinate movement* of flexion on the side stimulated, and extension on the opposite side takes place. *The movement* so initiated *persists*—cataleptoid state, due to persistence of reflex spinal and cerebellar impulses. When the posterior roots are cut, this cataleptoid condition immediately disappears; consequently the continuance of innervation which led to the cataleptoid condition, whether

spinal or cerebellar, or both, depended upon incoming stimuli by the posterior roots (Sherrington).

As the stimulus employed in this experiment was only temporary it is more reasonable to suppose that the persistance of the stimulus, which produced the continuance of the movement initiated by the cutaneous excitation, was due to incoming stimulus from the deep structures being put in a state of increased tension in the new position which the limb assumed. There is every reason why it should thus persist. The neurons transmitting the incoming currents from the contracted muscles are anatomically and functionally correlated with the neurons which transmit the outgoing currents; and the subcortical, spinal, and cerebellar nervous circles which are transmitting the most innervation currents will prevail. Probably this effect is mainly of cerebellar origin, for Horsley and Löwenthal showed that stimulation of the cerebellum in a decerebrate animal produced contraction of biceps and active relaxation of triceps. The uniform structure of the cerebellum suggests uniform function; probably it is an organ which functions as a whole. Hughlings Jackson has always considered it to be an organ which presides over continuing movement, and therefore static functions, as distinct from the cerebrum, which initiates successive movements.

Luciani's experiments show that total removal produces parasthenia, or defective force, paratonia, or defective tone, and astasia, or unsteadiness of contraction. The cerebellum is connected with the opposite cerebral cortex, and with the mesencephalon; it is probably to the severence of the connections with the latter, in ablation of the organ, that may be attributed the disturbances in gait and station.

In standing erect the cerebellum coordinates the innervation currents of the muscles of the limbs and trunk in such a way that the joints are fixed, and the line of the centre of gravity of the body passes through the knee- and ankle-joints. The weight of the body is therefore balanced upon the astragalus. This process is reflex and depends for its maintenance upon afferent impressions from the soles of the feet, and from the joints, muscles, and deep structures of the limbs and trunk, and from the semicircular canals.

In walking the cerebellum is reflexly coordinating the innervation currents to the muscles of the resting leg, while the cerebral cortex is coordinating the successive movements of the advancing leg in progression.

Incoordination.

An ataxic, on closing his eyes, manifests instability of station—Romberg symptom. Normally, equable reflex spinal and cerebellar tonic contractions fix the joints of the lower limbs, but when the afferent impressions from the deep structures of the limbs and from the soles of the feet are cut off by degeneration of the lumbo-sacral posterior roots, the ataxic patient has to rely upon impressions from the semicircular canals and visual sensations for the maintenance of balance, by tonic contraction of opposing groups of muscles. When he closes his eyes, owing to the absence of guiding sensations he is unable to voluntarily reinforce by attention this tonic contraction ; consequently, the source of compensation for maintaining equilibrium is removed, and swaying takes place, which, by the sensation from the semicircular canals, warns him of his danger of falling by the change in his state of consciousness of position, enabling him to recover himself by attention and an effort of the will. Romberg's symptom is an early phenomenon of tabes, and may exist without any cutaneous anæsthesia ; it is due to the interruption of the subcortical reflex spinal and cerebellar circles by degeneration of those neurons with coarse fibres from deep structures, *vide* fig. 48.

Before proceeding to discuss the mechanism of cortical coordination, it is necessary to make some observations concerning the efferent systems of neurons from cortex to muscle, by which voluntary reciprocal innervation takes place.

The psycho-motor neurons lie in small groups in the cortex in a series corresponding to the segmental spinal series, except that the lowest occupy the top of the ascending frontal convolution ; so that the longest axons arise from those cells which proceed to the lowest end of the cord (Sherrington.) These psycho-motor neurons become more

complex the higher we rise in the zoological scale; and the explanation is a proportional increase of combinations of association neurons which act directly upon an increased number of dendritic points of contact. Such cortical neurons are the effective agents of the will, regulating and adjusting by reciprocal innervation of spinal neurons the outgoing currents to muscles and the incoming currents connected with reflex muscular tonus. The diagram which has been used (fig. 48) is wrong in many respects; it represents, for simplicity, one psycho-motor neuron connected with *one* spinal motor neuron, whereas it is in indirect connection with many; otherwise the transection of the pyramids in the medulla should possess as many fibres as all the anterior roots put together.

Von Monakow is probably right in asserting that there is no *direct* connection between the psycho-motor neurons and the spinal motor neurons, but that intercalary neurons exist. Schäfer has shown that the pyramidal fibres end at the base of the posterior horn and not in the anterior horns. Sherrington has, however, found degenerated fibres proceeding to the anterior horns. Von Monakow in his recent work gives the following paths of cortical impulses :—

(1) A neuron, the axon of which enters the anterior horn and is brought into relation with groups of anterior horn-cells by means of an intervening intercalary neuron.

(2) A neuron which ends in a terminal arborisation in the mescencephalon, and is brought into relation by an intercalary neuron with another neuron, the axon of which passes down the cord to end at the base of the posterior horn, and is thus brought into relationship with the terminal arborisation of the afferent reflex spinal neurons, which again influence the spinal motor neurons by an intervening intercalary neuron.

The experiments of Sherrington show that there is reciprocal innervation of functionally correlated groups of antagonist muscles, and we may assume that a group of psycho-motor neurons, when physiologically stimulated, initiates in the spinal gray matter innervation currents which augment the contraction in certain groups—for example,

flexors—and inhibit the reflex tonus in the antagonists—for example, extensors. The adjustment of this reciprocal innervation determines the range, the rate, and the time of cessation of the movement; but subsidiary synergic groups of muscles nearly always co-operate, and are especially necessary in determining and maintaining the required direction of movement. Two paths from the cortex which Von Monakow figures may therefore be the means of regulating this reciprocal innervation. Thus, if volition initiates a flexor movement, an impulse proceeds direct to the appropriate flexor-motor neurons, and simultaneously an impulse flows to the spinal reflex anastomosis of the correlated extensor group, the result being increased innervation of the flexor group and inhibition of the tonus in the correlated extensor group.

If, then, we are correct in assuming that there is a tract from the cortex which regulates and adjusts reflex tonus, we can understand that, as this reflex tonus is progressively abolished by the destruction of the posterior roots, there is a disturbance in the balance of reciprocal cortical innervation of the spinal neurons, and the resulting incoordination is partly due to uncontrolled over-action of one system of cortical psycho-motor neurons.

The physiological stimulus which excites, under the influence of the will, appropriate groups of psycho-motor neurons for the regulation and adjustment of this reciprocal spinal innervation of synergic muscles, takes place at the synapses of their dendrites, with immediately correlated asscciation neurons. Voluntary reinforcement of this stimulus may take place by attention, which is a concentration of consciousness, so that there is a partial withdrawal of physiological activity from the cortex as a whole, with a corresponding concentration in a particular system of neurons comprising sensory terminals and correlated groups of association neurons, the result being a more vivid revival in consciousness of the perception which precedes the movement willed. There is a sense of effort attending this process, which we may assume is associated with a using up of potential.

After once the movement has been initiated, and repeated a number of times, the sense of effort becomes less and less, and it may be presumed that the nervous process is mainly subcortical after once the will has initiated the movement, and adjusted the necessary combinations of neurons for its performance.

In tabo-paralysis we have seen that a large proportion of the cases showed a degeneration of the crossed pyramidal tracts in the lumbo-sacral region, and we know that some of these cases showed very little ataxy in proportion to the degeneration, and a few exhibited less ataxy when the process of cortical degeneration occurred. The explanation may be that the long pyramidal fibres, having undergone decay, do not now overact and upset the balance between impulses going directly to the spinal motor neurons and those which influence reflex tonus.

No doubt the cortex is continually sending impulses down the spinal cord inhibiting reflex tonus; and this probably accounts for the fact that the knee-jerk may come back after cortical injury or disease, also for the existence of the knee-jerk on one side and its absence on the other. When the knee-jerk is present it means that some of the posterior roots of the lumbar enlargement are still present to conduct impulses, though not enough to allow of sufficient reflex tonus to give the knee-jerk; unless by a progressive decay, or acute degenerative changes, the cortical inhibitory influence is withdrawn; then (as in Westphal's experiments with strychnia), the knee-jerk is obtainable, because the afferent impressions now produce sufficient tonus. Jendrassik's method of reinforcement is probably explained in the same way, for by grasping the hands and pulling, attention is concentrated in the arm area of the cortex, and there is a withdrawal of physiological activity from the leg area, therefore cerebral inhibition.

The essential cause of locomotor ataxy is undoubtedly the degeneration of the posterior roots, but marked ataxy does not occur until there is some degeneration of the endogenous systems. This may be due to the fact that muscle-tonus may be reflexly induced by efferent impressions coming

from spinal segments other than those which are directly correlated with sensory and motor structures in which the posterior roots are completely degenerated.

Further observations which I have made upon monkeys, in which the posterior roots have been divided by Professor Halliburton, have convinced me of two facts: (1) That a true ataxy of *the upper limb* cannot be produced by division of posterior roots, as Hering has asserted; for either the animal showed no incoordination of movement; or if, in cases when there was an insufficiency of roots divided to produce paralysis, there were jerky, incoordinate movements, they did not persist more than a few days; indeed, the animal which Hering quotes from Sherrington's and my observations, as having shown incoordination, which it did only for a few days, was the one which we, some months later, showed at the Berne Physiological Congress, with complete sensory paralysis.

Jules Soury, in an interesting summary of tabes, refers to the fact that Sherrington and I had shown sensory paralysis, but it was left to the sagacity of Hering to demonstrate the importance these experiments had upon the pathology of ataxy. This important relationship I had been fully aware of from the time that I made experimental sections of posterior roots in monkeys at the Brown Institution more than twelve years ago; but I have not yet satisfied myself that section of posterior roots in animals can produce a condition similar to locomotor ataxy in man. Any incoordination of movement which may follow section of roots is either soon compensated, or followed by complete loss of voluntary power. We cannot produce that systemic, elective, and unequal destruction of root fibres artificially. Ataxy is due, not only to loss of function from neuronic destruction, but to physiological over-action of structures, which normally act in opposition to those which are destroyed.

One of the earliest paralytic signs of tabes is a loss of tonus in the dorsal flexors of the foot, and foot drop while the patient is lying on his back in bed (see Case 46). This may be partially due to gravity and the weight of the bed clothes. If the patient is told to flex his hip to the uttermost, it will

be observed that the synergic dorsal flexion of the foot with hip and knee does not take place. By the aid of vision and attention he can produce the flexion, but it involves continuous attention, and even then it is not synergic, but follows the hip-flexion. In walking the principal movement for progression is flexion of the hip, but the range of movement would be limited unless there was an associated flexion of the knee and dorsal flexion of the foot.

Such a patient requires a stick or two sticks to widen his base of support, in order that he may so adjust his body that the line of the centre of gravity may fall within the basis of support, and that he may look at his feet and guide their movements. Thus, by cortical processes involving continuous attention he compensates for the failure of the normal kinæsthetic sensations which are associated with every successive movement, and which in all movements which habit or practice have made semi-automatic and periodic, become sub-conscious. Why, normally then, does the sense of a false movement immediately evoke consciousness of it? Because volition, having determined the complexus of neurons to be employed in initiating a movement precise in range, rate, and direction, the successive combinations of neurons are by associative memory revived in orderly sequence—the complexus of sensations in the last movement sufficing for the revival of the next in the series.

Ingoing and outgoing currents are flowing through the cortical neurones without any expenditure of potential. A false step occasions a break in the lines of least resistance, with discharge of potential at the new arrival platform of kinæsthetic impressions. In both locomotor ataxy and general paralysis, there is a failure in coordination; but the cause is different; in the former, the coarse reflex and semi-automatic adjustments are at fault; in the latter, it is the fine cortical adjustment. In every conscious movement, both fine and coarse adjustment are used—the smaller the muscles, the more delicate are the minute alterations of their tensions, the greater and more varied is the complexity of their combinations and adjustments. Complexity and multiplicity of movements determine the number of spinal motor neurons

innervating groups of muscles, likewise the number and complexity of the communities and systems of neurons which precisely adjust the outflow of spinal innervation currents to the muscles. Take as examples the association of the hand and the eye, the ear, and the motor speech apparatus in the performance of the specially endowed human faculties, the communication of ideas by visual or verbal symbols, which brings directly into play successive combinations of systems of neurons too complex to analyse, but involving directly a large part of the cerebral cortex, the mesencephalon and spinal axis. However, the muscles which are especially innervated form but a very small fraction of the whole mass. The fine cerebral adjustment of coordination is continually and successively, by new combinations, infinite in number and variety, regulating the innervation currents of an infinite variety of spinal neurons, and thus adjusting the delicate tensions of correlated groups of minute muscles engaged in the expression of our thoughts and emotions. In general paralysis, the morbid process, early in the disease, strikes this fine adjustment in the cerebral cortex. The system of association neurons, which coordinate and associate the kinæsthetic impressions of near and distant related ideation centres with the psycho-motor neurons, are in a state of progressive decay, and although there may be no disease of the efferent path from the cortex in the early stage of the disease, yet, owing to defective and unequable stimulation of the cortical psycho-motor neurons, there is a failure in the precision of the reciprocal innervation of correlated groups of muscles used in verbal and written speech, manifest by tremor and incoordination. Some of the cases showed this very markedly, without proportional dementia and delusions. We must suppose that these were examples of functional selection in the morbid process.

The Physiognomy.

"There is no art to find the mind's construction in the face." One of the earliest signs of general paralysis is afforded by the expression, and we may recognise broadly

three types: (1) mask-like expression; (2) exaltation; (3) depression.

The first denotes a blank mind without active delusions of exaltation or depression, but often considerable dementia. Every emotion has its muscular concomitant in individual expression, and Darwin showed that so intimate was the relation between the two, that attention directed in such a way as to evoke the motor response of a particular emotion was accompanied by the correlated feeling.

In advanced dementia, the mind is a blank, there is absence of feeling, and consequently absence of motor response. Not only is there a blank expression in the face, but there is an expressionless hand. In those cases of the motor type, where there is lack of expression and marked speech affection without very much dementia, we may predict a more localised process (as in case 53) extensively destroying the neurons of association *immediately* connected with the psycho-motor neurons of the fronto-central regions. The conditions of exaltation and depression are, in all probability, partly due to neuronic destruction, partly to toxic and circulatory disturbances of the brain. The pathological process here acts like a magnifying glass, and control of what is passing in the mind no longer occurs. Like a muscular contracture in which one group of muscles continually overpowers the other, although the innervation current of the more powerful group is less than the normal, yet its effect is continuous and apparently greater, because the innervation of its reciprocal antagonist group is very much less than the normal.

The fixed ideas of exaltation or pain which we may consider as primary opposite emotional states, behave in the same way, and produce by the pathological process a magnified effect.

A patient not infrequently presents an expression of mental pain and dejection, but grandiose delusions and exaltation can be aroused in him by suggestion. The muscular concomitant is then shown by his facial expression; it immediately changes to one of exaltation, and the lustreless eye glistens when relating his wealth, strength, prowess or virility.

The emotions uncontrolled are reflected uncontrolled, and there is a failure in the reciprocal innervation of the muscles of expression owing to lack of reflex emotional tonus.

The subject of coordination is one of great interest, and I hope to deal more fully with its physiology and pathology in a future publication.

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* These works contain a very complete bibliography, and may be consulted for an analysis and summary of the conclusions arrived at by other workers.

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