General paresis / by Emil Kraepelin ; authorized English translation by J.W. Moore.

Nervous and Mental Disease Monograph Series No. 14

General Paresis

By
PROFESSOR EMIL KRAEPELIN
of Munich

Nervous and Mental Disease Publishing Co,
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1913

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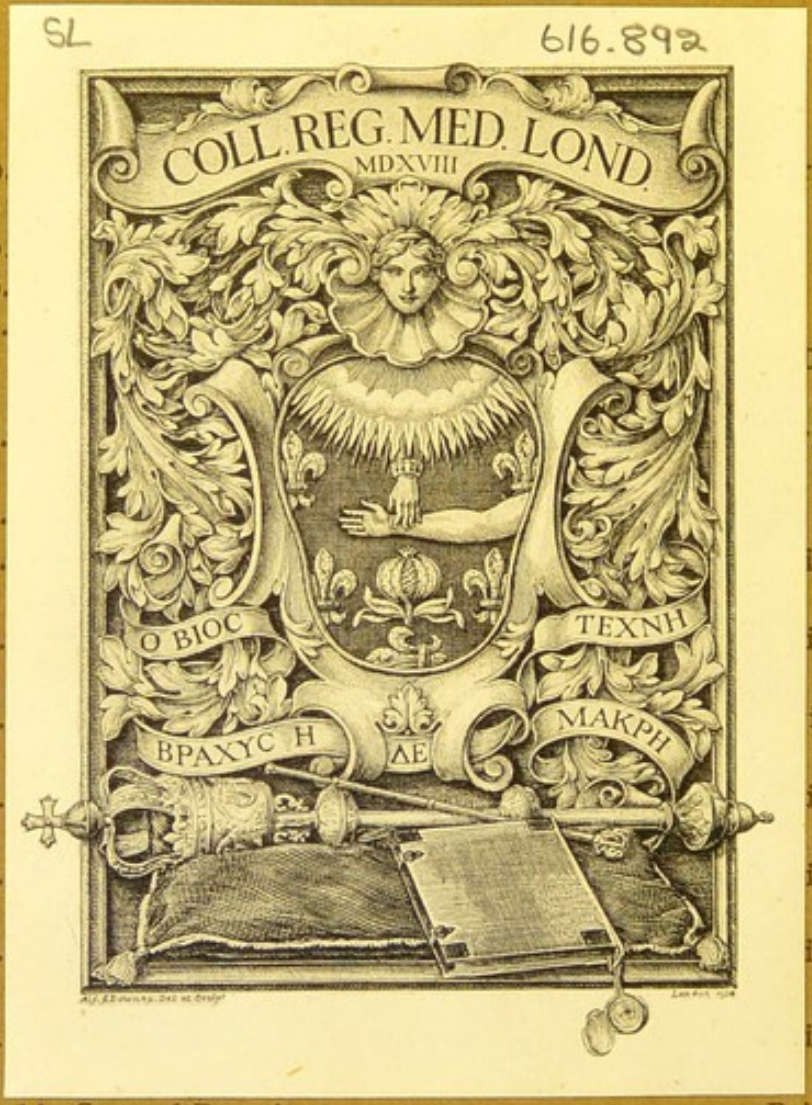

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SERIES, NO. 14

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Authorized English Translation by
J. W. MOORE, M.D

NEW YORK
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INTRODUCTION.

My "Introduction to Clinical Psychiatry" having up to this time been alone translated into English, colleagues Jelliffe and Moore have now rendered accessible to English-speaking physicians a chapter from my "Textbook of Psychiatry." This enterprise I hail with pleasure because, in precisely the domain here considered, the last decade has brought a series of extremely important additions to our knowledge, which are not yet included in the English edition of the "Introduction" more particularly. Although unfortunately we still find ourselves at a long distance from the solution of the paresis problem, nevertheless certain modes of enquiry and experimental possibilities are now available, arousing new anticipations in regard to a better understanding of paresis, and perhaps its improved treatment in the future.

Precisely America, so greatly our superior in the ample means at her command for the pursuit of learning, would find an extensive field of scientific research in the investigation of the etiology and body metabolism, the chemical and serological problems with which we are confronted. The long standing amicable relations between German and American psychiatrists will, I trust, receive an added support through the present translation.

E. KRAEPELIN.

MUNICH,
February 10, 1913.

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GENERAL PARESIS

CHAPTER I

General Symptomatology

The usual clinical picture of general paresis, dementia paralytica[1] or progressive paralysis of the insane ("softening of the brain") is a progressive deterioration leading to complete undermining of the whole mental and physical personality, accompanied by peculiar irritative and paralytic phenomena.

The earliest detailed and unmistakable description of this disease, which in its pronounced forms is particularly distinctive, seems to have been given about the end of the eighteenth century by Chiarugi and Haslam. Esquirol also undoubtedly recognized it when he wrote, "L'embarass de la parole est un signe mortel." However, the first clear presentation of the clinical relationship of the mental and physical symptoms was by Bayle (1822), Georget and Calmeil (1826) who also laid stress upon the disease alterations, especially the meningeal thickening.

It was a long time before the uniformity of this very characteristic combination of striking psychic and physical disturbances found general and undisputed recognizance. Views were

[1] Voisin, Traité de la paralysie générale des aliénés, 1879; Mendel, Die progressive Paralyse der Irren, 1880; Mickle, General Paralysis of the Insane, 2d ed., 1886; v. Krafft-Ebing, Nothnagels spezielle Pathologie u. Therapie, Bd. IX, 2; 2 Auflage von Obersteiner, 1908; Ilberg, Volkmanns klinische Vortrage, 168, 1896; Binswanger, Deutsche Klinik, VI, 2, 59, 1901; Chase, General Paresis, Practical and Clinical, 1902; Neumann, Die progressive Paralyse, 1906; Gaupp, Curschmanns Lehrbuch der Nervenkrankheiten, 619, 1909; Thomsen, Über die Bedeutung der Paralyse für die allgemenne Praxis, 1909; Joffroy et Mignon, La paralysie générale, 1910.

constantly reiterated which sought to give the paralytic feature a rôle quite separate from that of the remainder of the disease. Griesinger, in his text-book, took the standpoint that paresis was simply a combination of mental diseases, and even to-day we encounter, as a last echo of this gradually dying conception, the opinion that paresis is only a "syndrome." of very different disease-pictures. Nevertheless, it is to be emphasized that dementia paralytica, thanks chiefly to the anatomical discoveries but also to the studies in etiology and clinical features, of the last decades, now forms one of the best recognized diseases as to cause, symptoms, course, termination and post-mortem findings, which psychiatry, indeed even medicine itself, has produced.

General Symptomatology.—The distinctive feature of the mental disturbances which develop in the course of paresis is a *peculiar psychic weakness* which, at one time, dominates the disease picture, at another is accompanied by delusion-formation, alterations in mood, and various states of excitement and depression.

In the domain of the *intellect*[2] one of the first changes is often a marked difficulty in perceiving and understanding outward impressions,—a defect which is measurable even in its early stages. According to Ziehen the reaction time is markedly lengthened especially for differentiation and choice. The patient is absent-minded, inattentive, does not grasp events transpiring about him with accustomed clearness. He does not notice details, loses the drift in a conversation and fails to hear questions asked him or answers those directed to others. He mistakes persons and objects, overlooks important circumstances or changes, which would not have escaped him before; loses himself among familiar surroundings. I remember a man who suddenly, one day, was unable to find his place of work where he had been regularly employed. Wizel showed that the patients make many mistakes

[2] Förster und Gregor, Monatsschr. f. Psychiatrie, XXVI, 42.

in constructing simple figures with beans, from copy; they apparently only imperfectly comprehend the essential points. Heilbronner demonstrated that patients are often unable to make a number similar to 555555 using the numeral 7 or 3, because the proposition is not clear to them.

Even if the disorder at first glance seems not to have progressed far, it will often show itself in the inability to *fix the attention* for long periods. The patient has lost capacity for the longer and more involved thought-processes and the appreciation of finer allusions and witty remarks; he is not alert, loses the trend and connection. Thus, one can often make remarks about him in his presence without his knowing it. He leaves the physician, after his visit, with extravagant expressions of thanks, without realizing that he has received neither information nor advice concerning his illness. Finally, he may find it difficult or impossible to conduct himself properly even in the circle of his customary situations and obligations.

In this way, a more or less pronounced *clouding of consciousness* develops, and the patient lives now as in a dream or as if in a light state of drunkenness. One of my patients was held for intoxication by a police magistrate. Often this drowsiness which deprives the patient, to a certain degree, of his usefulness is an important diagnostic point in the beginning of the disease. Later, the disorientation in spite of apparent presence of mind, gives the impression, on casual observation, of an epileptic dream-state. The patient understands the questions put to him and his speech is fairly well ordered, but he has no idea where he is, with whom he speaks or what the situation is; he does not notice things in his surroundings but lives as though in another world. In the last stages of the disease the consciousness sinks steadily and finally to the lowest possible ebb, which precludes entirely the grasping and digesting of external impressions.

An *increased tendency to fatigue* is frequently among the first

symptoms of the disease. The patient finds his ordinary labor very difficult; he must frequently make renewed efforts, must rest, feels exhausted and disabled after a short period of activity. He hesitates at every small difficulty, becomes easily entangled and must often make a fresh start. Not infrequently it happens that he is overcome by weariness in the midst of his work or of a conversation, and falls asleep.

Sense deceptions usually play a slight rôle in paresis; so slight that their occurrence was formerly often denied. Nevertheless, definite illusions of the various senses are not infrequently observed. Obersteiner saw them in 10 per cent., which about corresponds with my experience, while Junius and Arndt record 17 per cent. Illusions of hearing seem to be somewhat more frequent than those of sight. Many times one deals with an habitual drinker, so that an association with an alcoholic disturbance must be considered. However, it appears further that auditory hallucinations can occur if the disease process affects especially the temporal lobes. Finally, also, under certain circumstances, an association with luetic endarteritis can come into question. Quite often one hears the patient, in a dissimulated voice answering his own remarks, so that a sort of dialogue takes place with God, the Kaiser or a relative, but without real hallucinations of hearing occurring. Rather, in this case, both the remark and the answer are uttered aloud by the patient while during a conversation with "voices" he either remains silent or only his answers to the hallucinatory perceptions are accessible to the listener. Serieux speaks of a patient who felt speaking in his tongue while he ground his teeth together. Hallucinations of touch with ideas of mystical influence occur frequently. Now and then one also observes hallucinations of smell. Especially prominent visual hallucinations occur in patients with optic nerve atrophy. They may be so vivid that the patients do not notice their blindness but think they move in a world of variegated, highly-colored visual

impressions. "I can see in the dark," a patient answered me, irritated by my question whether he perceived anything; he had long lost all power of vision. Another thought himself in an "uninhabited region"; he prayed for new eyes.

The disorder which affects *retention* and *memory* is usually a profound one, so that the disturbance in this field may be regarded as diagnostically especially important for paresis. To be sure they are, in the first place, not nearly so severe as in Korsakow's psychosis, as Ranschburg was able to show by experiment, and a certain improvement is noticeable with practice. Gregor found in his memory experiments a relatively good retention power, difficulty in association, lessened capacity for learning and marked disturbance of attention. Perhaps in the beginning it is the uncertain and dreamily vague perception of outward impressions which permits them to be retained in memory for only a short time.

Hence the patient forgets, unlike the ordinary memory relations, the *more recent happenings*. He does not know what occurred to him a week before, with whom he took a stroll yesterday, what letters and what work he had to attend to. Later the disorder steadily increases and the increase is usually hand in hand with the general deterioration, although one often encounters a patient who, in spite of fairly preserved mental alertness, cannot remember what he did a quarter of an hour before, whether he has ever seen the physician who has greeted him every day. One of my patients after many months in the hospital inquired of me every day for weeks where he really was,—he must have slept, had just awakened and found himself among strange surroundings. After a half-hour he had again forgotten the information given him and was constantly astonished at the changes which must have been made "during his sleep." Others live so completely in the present moment that they never know the time of day, whether it has been a long or short time since

arising, whether or not they have eaten dinner. They undress in the morning thinking it bedtime, are irritated in the evening because their coffee is not brought.

Such serious disturbances, it must be admitted, are only present in far advanced cases but even in the very beginning they are often sufficiently prominent to enable one to recognize the existence of paresis with great probability. The consciousness of this uncertainty and forgetfulness sometimes leads the patients to make records immediately of every little occurrence, every idea which they have, which notes, however, they are themselves unable to comprehend at a later date. Memories of the remote past, on the contrary, are retained in complete form for long periods, while fresh incidents are quickly and totally lost. Thus many patients with serious loss of retentive power may still be able to reckon with figures or to play cards fairly well. Paretic women often give their maiden names when asked, while they recall their married names only with difficulty or not at all.

The paretic loses very quickly the *time relations of his memories.* Since his perceptions are not brought together in that close-linked chain of memory pictures which enables us, in reviewing, to measure the duration of interval between each happening and the present, he is unable to arrange the past into definite periods of time. He cannot recall the sequence of occurrences nor their relationship with one another. Time limits melt into each other or are effaced. He makes gross errors even in a simple test such as the going over of a short limited period. Thus, he does not know whether months, weeks or days have passed since a certain event, as his admission to the institution.

Even in the earliest stages of the disease it is very characteristic to find an inability to adjust statements of time to each other in their proper relations. Although he may give the year of marriage and the age of his oldest child, he may still be unable to reckon how long he has been married, how old he was at the

time of his wedding or when the child was born. Often under cross-questioning in this line he falls into wide discrepancies without noticing it. Even the helpless glance which he casts about, the hesitating manner, or the evasive answer that it is all recorded in the family records—that the doctor himself knows, all go to show the profound incapacity of the patient. Eventually he does not know the day of the week, the date and often, not even the year, "because he has no calendar," or he allows himself to be easily drawn into errors in his answers. For example, he often gives the year and day of his birth for the present date and can be misled into giving such impossibilities as "February thirtieth." He does not make use, as would a normal person, of such aids as the appearance of the ground, the position of the sun, the light, temperature, etc., because he does not know how to turn them to account. In spite of the hot stove he thinks it is summer and fresh cherries on the table do not make him doubt that the month is December.

Besides the more recent impressions, the memory disturbance always involves, in the course of time, the more remote past. The patient can no longer give a connected account of the principal events of his life, confuses all the happenings, mixes the names of his acquaintances. Gradually he loses the knowledge which he has gained in school and in business. He becomes unsafe in his grasp of the sphere of ideas which form the content of his daily occupation, makes mistakes in the employment of a foreign language in which he was previously fluent, cannot recall to mind geographical and historical facts of which he was formerly complete master. An especially marked and often important sign is the early appearing difficulty in calculating. As a rule the mechanically-learned multiplication-tables are relatively long retained but even here mistakes are early noticeable. Errors are soon detected in those calculations which require the carrying of numbers in the memory, namely subtraction with a borrower (73–15) and division.

The continuous and progressive *deprivation of the store of ideas* leads eventually to complete loss of the mental possessions. Naturally the rapidity with which this process is enacted is very variable. It depends first upon the severity of the disease, then also upon the personal ability and power of resistance of the individual. The order in which the mental powers are lost depends essentially upon the resistance afforded by each element. Much used thought associations resist longest. The more frequently recalled day of birth or marriage is retained more easily than the accompanying year, and will therefore often be given alone in answer to a question. The merchant loses ability to give the multiplication tables later than the farmer; a peddler will easily reckon small sums in money even when badly deteriorated. Occasionally a single impression accidentally brought into the foreground through some incident will be retained remarkably long. An already deteriorated patient repeated constantly for years the number of his room in a water-cure sanitarium in which he had been until brought to the clinic. In the last stages of the disease the patient can no longer tell whether he is married, what his children's names are or whether he has any children, what his business is and perhaps forgets his age, his residence and even his name. Occasionally one can, for a short space of time, obtain surprisingly correct answers,—an indication that the memory pictures are not lost but only that the patient has become unable to recall them. Lack of comprehension often plays an important rôle, and may be helped by a suggestion, At the end, the ability to remember sinks literally to nil; the patient may not recognize his nearest relative.

In some cases, besides the diffuse weakness of memory, circumscribed gaps may be found, which are especially apt to correspond to paretic attacks. One of my patients, after recovering from a brief state of confusional excitement, had lost all recollection of the previous five months, although she had fallen in love

and married during that time. While she was quite clear in other respects, she was much shocked when her husband appeared. After another similar attack which occurred later she could not remember her first hospital residence of six weeks which had been only shortly before, nor could she, although at the time perfectly clear, recognize the physicians and attendants.

Very frequently the lapses of memory are filled out of the imagination. Not only are dreams and stories which have been heard and read brought into their own past but also a number of independent ideas as they arise are thus employed. The patient has experienced fabulous adventures, fought great battles, occupies a confidential position with numerous celebrities, has taken part in historical events for unthinkable ages. He has aroused England, wiped out Persia, stolen thousands of the most beautiful women, invented a system of arithmetic and a process of changing wood to gold by electricity, composed the poems of Hafiz, discovered America with the Vikings. In this way he falls into a peculiar highly-colored play of the most fantastic ideas of a kind which belong to our dream-life and are in marked contrast to his otherwise fairly well-ordered mind. Such dream-like, hazy states with confabulation appear in their most exaggerated form in patients with optic nerve atrophy; they may last months and years. On the other hand we often observe that remembrances of recent events are falsified by more remote reminiscences. The patient tells that a half-hour before, he received a communication, a letter, or paid a visit; that he dined yesterday with the Kaiser; has fallen in love, that morning, with a princess; has taken a journey. At first such stories may seem simple boasting or intentional lying, with the view, for example, of obtaining his release, but the patient usually "talks himself" into believing them. As a rule one can by suggestive questions bring out and influence such stories. During this process one notes that the patient at first feels somewhat uncertain about these utterances that have

been drawn from him but keeps calling more and more upon his imagination.

This susceptibility of the memory to outer influences is only a part of the general pliability of the thought of our patients. The ideas which they conceive do not take fast root and do not exert any influence on the further thought and action. They are quickly replaced by outward impressions or new, immediately arising thoughts and moods. The patient is in a high degree distractible; an accidental interruption suffices to divert him to an entirely new track. His train of thought is therefore flighty, aimless and confused. Many times a great monotony is noticeable; the mentally impoverished patients continually bring forward the few remaining ideas at their command because they have forgotten that they were uttered before and also because they are unable to suppress what once comes into their fancy.

Another important and early appearing symptom in the realm of the intellect is the *loss of judgment* of the paretic. To the experienced this symptom reveals the whole extent and severity of the disease, although there may still seem to be no ground for anxiety. As Förster and Gregor have shown, patients, even in simple psychic tests, do not notice contradictions and unclearness in their thoughts. In doing problems they are distracted by the sound of the numerals ($9 \times 9 = 99$), or thoughtlessly give any number that enters their head. In reading they do not notice omissions, the skipping of a line, gross distortions; they pay no attention to the sense nor do they attempt to correct errors. They repeat the content very incompletely with alterations and omissions of important parts, but do not realize its senselessness. They arrive at only a very incomplete understanding of their situation and of things going on about them. The complacency with which they bring forward some absurd plan, the easy disposal of obvious objections, the weakened stand against deluded notions, the inability to pursue an ordered train of thought, the rash conclusions,

all attract attention at an early period, although persistent, well-learned habits of thought may still conceal to a certain degree the extent of the disability in the highest mental processes. The patient gradually loses his grasp on those fundamental principles by which we judge the world, as well as the ability, through observation of facts, to apply a proper critique to the creations of his imaginations. He finds himself in a dream-world in which all his own ideas, his own wishes, his own fears, are in keeping. In this way he arrives at the formation of delusions. His whole environment, all his associations, are altered to his view because he sees them with different eyes, and is unable to note the incompatibility of his mistaken impressions with the facts.

It is the *basic intellectual weakness* that gives paresis from the beginning its peculiar stamp. Relatively seldom we observe, for short or long periods, a connected delusional formation which even then is characterized by a certain vagueness and changeability. Usually the most diverse ideas are entangled one with another without regard to the most obvious discrepancies. Thus the senselessness and fantastic nature of the paretic delusions are apt to carry them, with disarming candor, far beyond the limits of probability or even possibility. Where the activity of the imagination overcomes the nullifying power of criticism, the wealth and exuberance of the delusional formation is enormous.

Another fact, dependent upon the mental weakness of the paretic, is that the delusions are not fixed but are continually altered by the influence of internal and external stimuli. New ones are brought up and the old fall into the background. Every expression of the paretic's delusions shows numerous and essential differences from those previously uttered. The erstwhile count has perhaps become Kaiser and immediately afterward is the youngest lieutenant. Indeed one can often, by insidious questions and persuasion, cause the patient, in the course of a few moments, to expand his ideas to the most monstrous proportions.

On the other hand, we often see the most exaggerated delusional formations suddenly disappear. They are simply forgotten, without the substitution of new ideas. They are seldom corrected with full recognition of their falsity.

Paretics seldom have a true realization of their condition. On the contrary, the patients frequently feel healthier than previously or, at least, they do not appreciate that they have lost all their mental powers. This is simply because they have not the ability to compare their present condition with their long-forgotten normal days. Only in the beginning of the disease is there sometimes an understanding of the nature of the affection and their eventual fate. I have a letter from a colonel in the army in which he declares his intention of taking his life because he has softening of the brain and must become a silly idiot. The further course of the disease bore out his fears only too well. Another officer shot himself and his fiancee because he felt this disease coming upon him; the autopsy showed that he was right. It is not very seldom that patients experience a distinct omen of a serious and incurable disease. Many feel really ill on account of a variety of nervous symptoms, or reveal true hypochondriacal complaints without grasping the real significance of their disease. Many patients believe that others about them, for example the wife or the family physician, are insane and they agree readily to a proposition for a consultation with an insanity specialist.

The patient's *disposition* is hardly less affected than his intelligence. In the earlier stages of paresis it is usually the increased excitability which attracts attention. The patient is capricious, easily angered and surly, thrown into transitory states of emotional excitement by trivial causes, at which times he completely loses control of himself and flies into a violent passion. On the other hand a certain insensibility to higher emotional strains is to be noted which points to diminution in the deeper and finer feelings. The patient is careless in the face of threatening

danger, takes little heed of approaching difficulties, is undisturbed by severe misfortune, is quickly reassured. The pleasure of mental exercise, the enjoyment of art, the satisfaction of genial relations with environment and with family, give way to a careless indifference which is in striking contrast to the excitability otherwise manifested by the patient.

In the further course of the disease the excitability which was present at first gradually disappears while the lack of deeper, lasting feeling becomes still more marked. The coloring of his mood agrees usually with the content of his delusions. Ideas of grandeur are accompanied by a contented, often very happy mood, while we observe deep depression or states of great anxiety with the harassing delusions. Occasionally, it is true, gloomy delusions can be present with a cheerful mien. It is not the rule, however, for the whole course of the disease to be accompanied throughout by the same general mood-tone. On the contrary, the abrupt alteration of the emotions is so highly characteristic of paresis that recognition of the disease can depend entirely upon it. In the midst of happiness there is a sudden storm of tears or the hypochondriacal distress may be lost in the childish delight over some unexpected diversion. It is especially remarkable that it is frequently possible to artificially induce these quick changes, by the mentioning of suitable ideas, indeed even by the tone of the voice and the facial expression. A kind of silly elation or angry discontent can, without dependence upon delusions, accompany even the deepest degree of emotional dullness.

Naturally the *character* of the patient is materially altered by these disturbances. In place of the earlier stability and independence occurs an increasing loss of will-power, which manifests itself in marked irresoluteness, indecision and instabilbility, frequently also in foolish, headstrong waywardness. As his own voluntary activity, his "initiative," is reduced the patient can almost always be led in any direction by adroit handling. He is

credulous and trustful and therefore falls victim to the clumsiest frauds and extortions. His transfer to an institution, which is so dreaded by the relatives, is often accomplished without difficulty, much to their surprise. The careless, matter-of-fact way in which the paretic, although without any feeling of illness, allows himself to be placed in a "cure" and to take up his abode in an institution, the way he praises the beautiful room, the excellent care and treatment and his willingness "to stay awhile," show perhaps most clearly the patient's weakness of will. A few friendly words, a joke, an evasive reply will always pacify the patient who daily sets "to-morrow" as the day of his departure by the night express, who insists that he cannot be spared at home, that his plans for the future are being upset. He is quite incapable of any orderly work, since he either forgets or neglects his obligations, or performs them in a disorderly manner with omissions and errors, or he busies himself with aimless things. A very thoughtful and cultured man on his entrance to the clinic asked for an encyclopedia to read from and on the next day requested a new volume as he had read the first through.

On the other hand the patient is wont to follow without heed any impulse or whim that may occur to him. His actions bear the stamp of thoughtlessness and lack of method. One of my patients sprang from a second-story window after a cigar stump which he had noticed and sustained a fracture of the fibula; another tried to let himself down by a very thin thread and fell to the ground. Even crimes, such as stealing, may be committed without the patient being able to appreciate the significance and consequence of his acts. Frequently there is a sudden exaggeration of business activity which consists more of making plans and getting started than in actual accomplishments. In quick succession and without consideration the patient makes the preliminary moves, so soon as the thought occurs to him, only to forget and drop them the next moment for something larger.

In the patient's *behavior* there is a noticeable bluntness to the demands of propriety and custom which leads him, like one who is intoxicated, to exhibitions of tactlessness, lack of restraint and even gross offenses, without his having the least realization thereof. Those inculcated finer restraints and impulses which regulate the outer form of our conduct according to our surroundings, are usually very early lost in paresis. The patients are careless of appearance, have no sense of order and cleanliness, go about in neglected dress and with soiled finger-nails, form friendships with strange men, boast obtrusively, disclose their most intimate affairs. They forget the deference due those in higher station, force themselves into prominence, depart without farewell, come too late, leave their guests in the lurch, hold uncalled-for and unseasonable conversations without having the least consciousness of the painful sensations which they cause. Where a long habit or natural instinct has impressed itself very deeply upon the person's character, one can recognize the shadow of the old forms even in a much deteriorated patient.

The frequently occurring sexual excitement manifests itself in the telling of lewd stories, the seeking of doubtful companions and shameless debauchery. As deterioration progresses the conduct of the patients gives more and more the impression of coarseness and unrestraint. They submit cheerfully to any rebuke, break into the conversation of strangers, attend to the calls of nature regardless of the surroundings. They fall greedily upon anything in the way of food, help themselves to whatever comes to their hand, engage at once in a fight if angered. Still later even this excitability is lost; instinctive picking and rubbing, grinding of the teeth and occasional inarticulate mumbling are the last psychomotor manifestations which the increasing paralysis allows.

Finally, there sometimes occur in paresis some of the disturbances of will which are spoken of as catatonic. Not only is catalepsy, at least transitory, frequent enough, but also echolalia,

echopraxia and verbigeration, the continuous almost rhythmical repetition of the same sentence, word or syllable. A patient repeated incessantly the words "jacket, heath, glycerine." Indications of senseless resistiveness are many times observed, such as mutism, refusal of food, objection to commands or interference, assuming and holding peculiar positions, retention of urine and feces. From one patient's bladder we removed three liters of urine. The resistiveness is, however, much more variable and inconstant than in catatonia and may often be overcome by persuasion, so that a doubt remains whether the outwardly similar symptoms have the same underlying cause. Often the patient who has been mute and resistive suddenly begins to speak and act normally. One often observes spitting, grimacing, mannerisms, occasional stereotyped movements, dancing about, ceaseless rubbing and picking, grunting, smacking, chewing, choking, defensive motions, distortions of the body, oscillatory movements, monotonous, long-continued cries. In the case of these, also, it is doubtful to me whether they are identical with the similar catatonic symptoms. Many of the stereotypies, even the teeth-grinding and choreic movements appear to be more of spasmodic origin, while others again have the appearance of the abortive remains of originally sensible acts. I have not, up to the present, been convinced of the justification of separating a special catatonic form of paresis.

The *neurological disturbances* which occur throughout the course, are what, more than anything else, give the particular clinical stamp to the disease.

A common symptom at the beginning is severe *headache*. This is usually a dull but extremely heavy pressure as if the brain were compressed by a great weight. It is generally most severe in the frontal region. Associated with this are indications of circulatory disturbances (ear-noises, sparks before the eyes, dizzy feeling). On the part of the *sense-organs* there is

often, in the beginning, a hyper-acuteness, later more or less blunting of sensibility, which is probably due to the psychic state, especially the diminished attention. A characteristic visual disturbance which frequently follows paralytic attacks is described by Fürstner and attributed to focal disease of the occipital cortex. It consists in a difficulty in recognizing and localizing objects, although the eye-grounds are normal. Word-deafness and asymbolia are also often observed. Now and then hemianopic disorders are noted, especially after paralytic attacks. On the other hand, actual disorders of the eye itself appear. Atrophy of the optic nerve of various extent is observed in 4–5 per cent., according to Mölis, in 12 per cent., especially in those cases associated with posterior column affections. It is sometimes the first indication of the approaching disease. Besides this we occasionally encounter a, by no means peculiar, "retinitis paralytica" and a number of other less frequent and very various changes in the eye. Keraval and Raviart saw retinal changes, mostly of a slight nature, in 82 per cent. of the cases, Joffroy only in 12 per cent. De Martins found frequent disorders of smell and taste, particularly an almost constant loss of ability to recognize the taste of salt; Toulouse detected insensibility to the smell of camphor in a third of his cases.

The symptoms referable to *cutaneous sensibility* are very striking. In the beginning of the disease there are a variety of unpleasant sensations, itching, burning, tearing, pulling, "rheumatoid" pains, which in some cases are for a long time the only signs of the disease. Where they occur in the girdle region one has to think of tabetic disturbances. In one of my patients the disease began with neuralgia of the penis and testicles. In a few cases there is an especial sensitiveness to cold. Occasionally there seem to be hallucinatory experiences in the sphere of touch sense. They feel electricity, think that someone is in bed with them. It is difficult to say what part is played in this by the very common hypochondriacal delusions with bodily sensations.

In the further course of the disease there always develops, sooner or later, a decided diminution in all the qualities of skin sensation, but more especially a marked hypalgesia. Even in the relatively early stages, if one diverts the patient's attention by questions it is often possible to stick a pin through the skin without his knowing it and to his great surprise. Pilcz has found that a strip of skin about the neck, a girdle about the buttocks and the posterior surface of the thigh are as a rule not anesthetic. The internal portions of the body, however, appear to lose their sensibility. Usually painful diseases of the stomach, intestine, pleura, internal genitalia, or an over-filled bladder cause the patients no discomfort. This insensibility to pain favors the occurrence of all kinds of injuries, especially burns, because the patient, not appreciating the danger, does not guard against it. The patients pick their finger-wounds, bite their finger-nails to the base, stick themselves recklessly in the mouth. I know an army captain who, during the night, lacerated his hand with his teeth because it seemed to him to be something which did not belong to him. Insensitiveness of the ulnar nerve at the elbow (Biernacki's sign) appears to be frequent.

The *motor symptoms* are especially prominent in paresis. More or less outspoken indications of apraxia are common, which indeed may be mistaken for simple clumsiness. There is an inability to carry out several different movements in quick succession (Babinski's adiadochokinesis), such as to close the eyes, open the mouth and show the teeth. The patients fall into errors very easily and confuse the order of the different steps in the formula. In complicated actions, long pauses are interposed between the parts because the patient has lost the thread or he gets on the wrong track entirely. Often when they receive the request they first go through awkward, senseless movements. They act at random without a clear purpose. Definite motor apraxia with inability to imitate movements is not at all rare

after convulsive attacks, but is usually hard to prove. Like other observers, I have been able to follow the symptom definitely for several months.

The carrying out of isolated movements meets with difficulty and delay. The gross strength is fairly well preserved, except for the weakness following an attack, until the increasing paralysis gradually destroys it. The finer adjustment of muscular effort suffers early; ataxia develops. The movements are clumsy, slow and awkward. The patient cannot catch a quickly moving object, button the coat, thread a needle or knit. Occasionally intention-tremors occur. More frequently there is a very characteristic preservation of motor impulses; the patient presses the proffered hand violently and persistently without releasing it. The gait is unsteady with a wide base and is shuffling, often spastic; at times, especially before an attack, the patient leans far over to one side.

The features are flabby (flattening of the naso-labial folds) and expressionless. Often one sees a difference in the two sides, drooping of one corner of the mouth, weakness on special effort. Fibrillary tremors and by-play of other muscles during mimicry are extremely common; one sees a sort of "heat lightning" waves through all the facial muscles. The whole carriage of the body is lax and without elasticity. One recognizes these disorders in the dull, silly expressions of the faces in the group picture (Fig. 1). The patient in the middle shows his exalted mood in the bouquet he wears, his neighbor to the left has a left-sided facial paresis.

The voice is monotonous, sometimes tremulous; it loses its capability of expression and often its normal resonance (paresis of the vocal chords), in some cases the first symptom of paresis, namely in singers. The tongue often deviates, shows strong fibrillary tremors; it is protruded awkwardly, jerkily and with many associated movements such as a wide opening of the eyes,

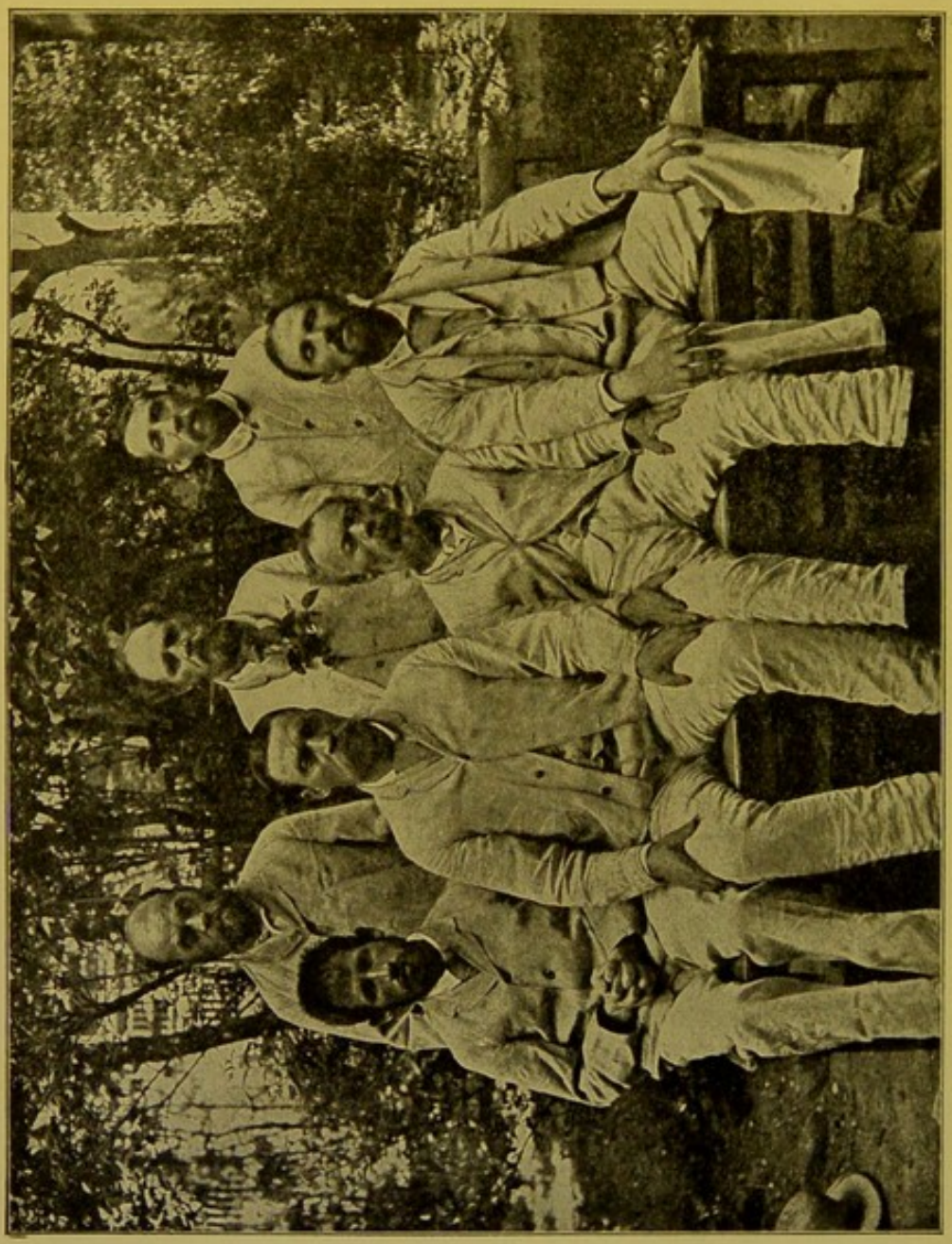


FIG. 1. Group of Paretics.

wrinkling of the forehead; sometimes even the assistance of the fingers is required. When it is extended the patient often involuntarily clasps it with the teeth to prevent the jerking. Swallowing is difficult especially in the later stages. The patient chokes easily but by reason of the insensibility of the laryngeal opening the reaction is not always sufficient. A further indication of bulbar disease is the frequently-observed forced laughing. "I have no reason for laughing," said such a patient to me. In another case, besides the general paretic changes, a large gumma was found in the pons. Frequently one observes in advanced deterioration, long-continued, rhythmic grinding of the teeth, which can be regarded as almost pathognomonic of paresis. In one case I saw the disease ushered in by a severe accessorius spasm. In a few instances writer's cramp occurred, in others rhythmical facial tremors, once spasmodic winking and wrinkling of the forehead. There can also be choreic movements. Where these antedate the outbreak of the disease by a long time there is always a possibility of Huntington's chorea to be considered.

Among the most important signs of paresis is the change shown in the *speech*.[3] We have to differentiate between aphasic disorders and those of articulation. Transitory, brief aphasia occurs very frequently in connection with paralytic attacks. One of my patients was for weeks unable to name a single object shown him, although he recognized them at once. Paraphasia is apt to be much more severe and may exist for several months. In this disorder the patient either calls things by their wrong names or certain stereotyped expressions constantly recur in designating objects. "I have two meters," one of my patients used to say happily as he drew a handkerchief from each side pocket; "I have the pearl-white stones." Perseveration is frequently observed. Much less common is word deafness, which may be difficult to recognize on account of the mental enfeeble-

[3] Trömner, Arch. f. Psychiatrie, XXVIII, 190.

ment of the patient. But one often notices, especially after paralytic attacks, that even the simplest remarks are not understood, although commands given by gesture are obeyed at once. Closely associated with this disorder is the loss of musical gifts, of the ability to catch a melody and especially to sing and to repeat a song.

Among the central speech-disorders must also be grouped the occasionally-observed agrammatism, the inability to form sentences correctly. The patients speak like children, without conjunctions or in the infinitive. Far more frequent than this is the difficulty in composing words from their syllables. According to Trömner we should distinguish between the dropping out of syllables ("electrity"), contraction ("exity"), and reduplication of syllables ("electricicity"). This last form which depends upon the involuntary addition of an unaccented syllable, occurs usually at the termination of a word. The final syllable is often repeated, in spite of the obvious efforts of the patient, three, four or more times before the vocal organs come to rest ("Anton-ton-ton-ton"). For this very striking symptom, which corresponds to similar affections in other muscle-groups and is to be classed with the perseveration disorders, I may suggest the term "logoclonia." As a result of all these disturbances, which are often accompanied also by aphasia, the speech may be reduced to a mixture of senseless, frequently-repeated combinations of syllables. I know a very well educated patient in whom the first paretic symptom noted was a slight apoplectic attack after which, for a few hours, he jumbled together unintelligibly the five or six languages of which he had command.

Disturbances in *articulation* are still more frequent than central speech-disorders. They are perhaps first noticed only after paretic attacks or in excitement but later are continuously present. They are separable into two distinct varieties which are usually intermingled in each case—the paretic and the ataxic coördination

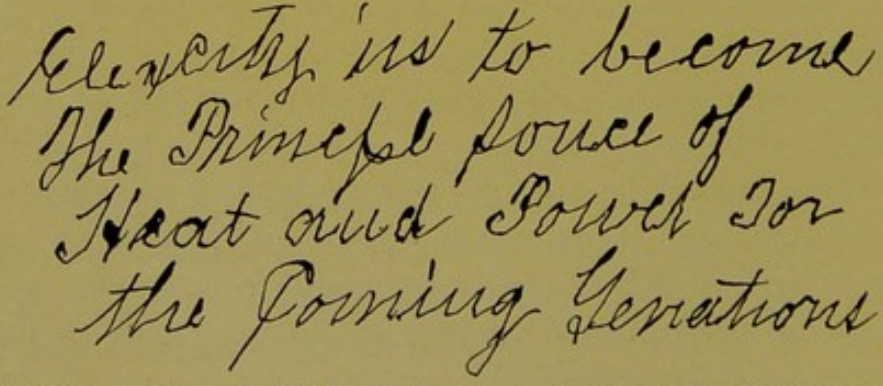
disorders. The difficulty in movements of the lip and tongue muscles prevents the patient from pronouncing the individual letters clearly and still more from speaking complicated combinations of letters in correct sequence; one letter or syllable slurs imperceptibly into another. There is thus a drawling of the speech, frequent stopping (hesitating), sometimes also marked pauses between the syllables, generally with loss of melody and of correct meter (scanning). The difficulty in speech is often made very noticeable by the active associated movements of all the other face muscles. The forehead is wrinkled, the brows puckered, the eyes narrowed and there are many movements of the cheeks.

At the same time, the speech, especially when continuous, is indistinct and hardly intelligible by reason of the slurring over of the poorly articulated sound combinations (slurring, lisping speech). Since it belongs to the bulbar speech defects it may be looked for in alterations in the medulla, especially in the region of the facial and hypoglossal. It is nearly always accompanied by monotony of speech from failure of those inflections which, in natural discourse, give strength and pitch to the voice sounds. Further, the arrangement of the sounds into syllables is affected, —a symptom which, together with the above-mentioned difficulty in grouping the syllables into words, is called "syllable stumbling." Inconvenient transitions of sound are replaced by easier ("Epistiple" instead of "Episcopal") or simple shortening or simplifying ("vegibles," "elecric"). Many times the formation of a syllable shows the influence of previous or following syllables and letters or of neighboring words, just as in an ordinary slip of the tongue ("truly ruly" for "truly rural," "brigrade" for "brigade"). The patient is usually not conscious of this difficulty or refers it to some circumstance such as that he is thirsty, his mouth is dry, the food is not nourishing enough, that the test gets him so excited.

The few cases in which we took records of the vocal vibrations showed many disturbances, of which the poor differentiation of the successive speech movements seemed to me especially striking. While, in health, the vowel vibrations and the consonant movements appear to be sharply separated from one another, in paresis they often overlap; the vowel sound is sustained while the consonant is beginning, or it begins before the other is finished. This fine distinction which cannot be detected by the ear, is in fact the first beginning of ataxia of speech.

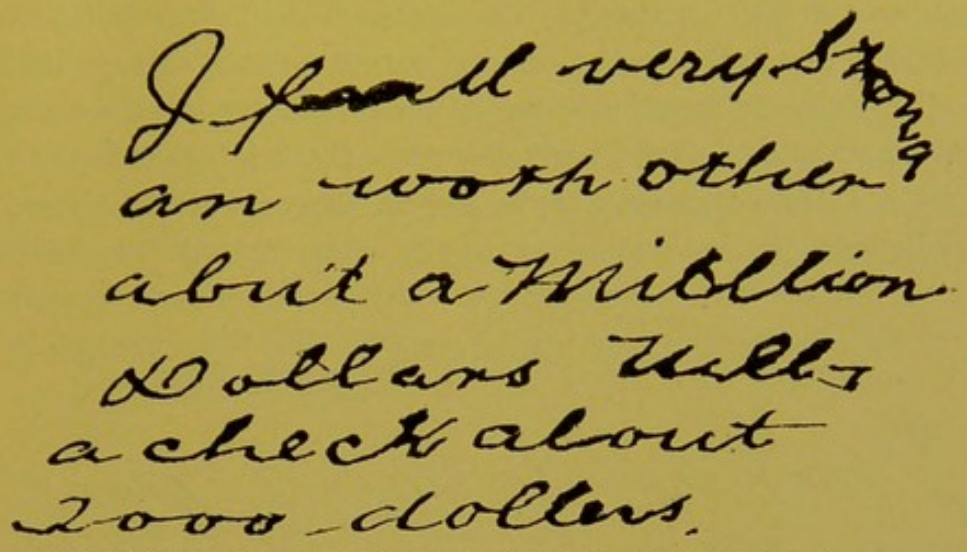
The central and ataxic speech disturbances are best shown, as Rieger has determined, by reading aloud. In this test the patient, on repeated trials, keeps forming different combinations of words and syllables which only fragmentarily approach a resemblance to the text. He thinks he has read correctly although he has not understood the content. It is not now possible to determine how far the sensory perception of the text, how far the association of the word-image with its concept on the one hand and with the concept of speech-movement on the other, how far, finally, the interplay of impulses, is concerned in the ultimate development of the disorder.

The *writing* shows alterations very similar to those of speech. The individual movements are irregular and uncertain but do not show the uniform fine, wavy lines of the senile tremor; the strokes are frequently much longer than they should be. Corre-



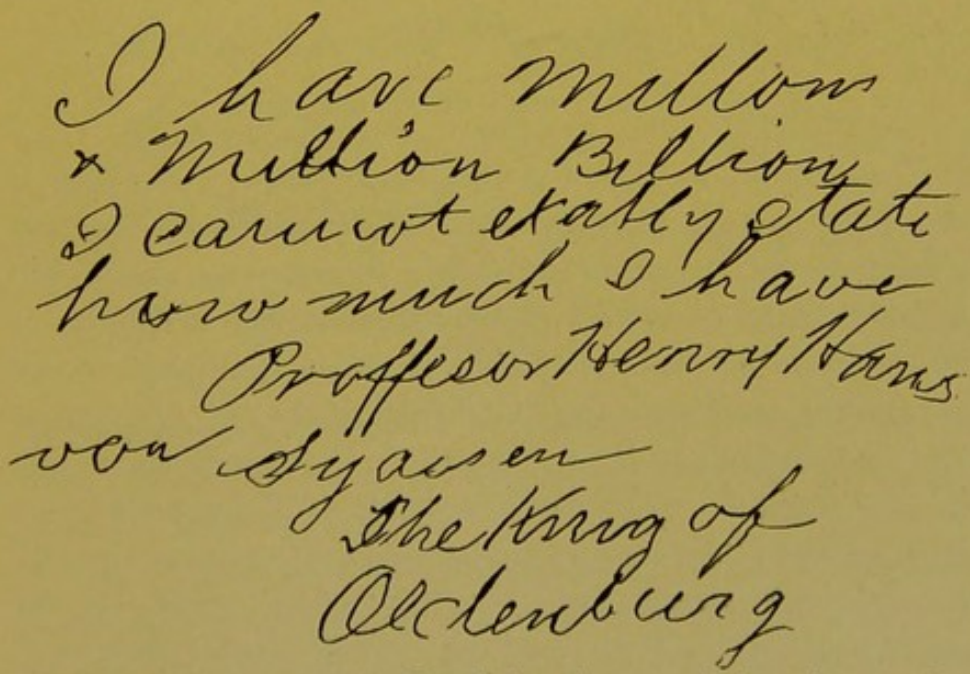
Writing specimen 1. Moderate tremor, omissions and reduplications.

sponding to the syllable-stumbling of the speech disorder, occur misplacements, omissions and repetitions of letters and syllables. Of the samples of writing given, the first is by a man of con-



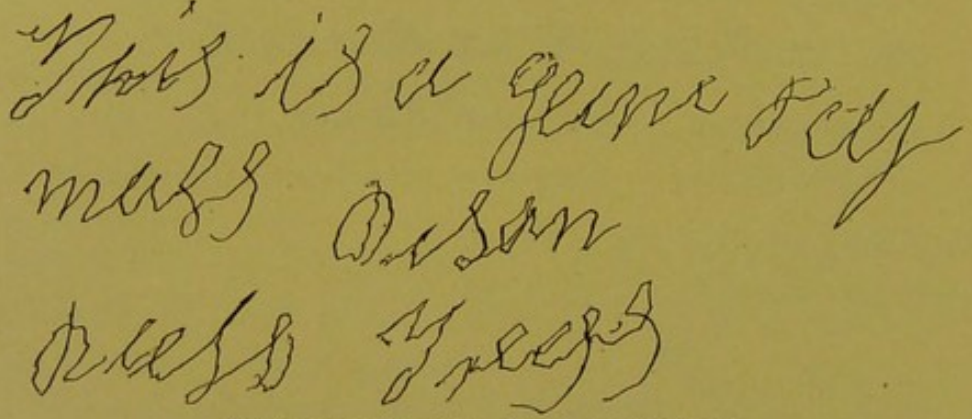
Writing specimen 2. Slight ataxia; omissions, perseveration.

siderable intelligence suffering from tabetic paresis. The arrangement and construction of the letters is fairly good, but the lines



Writing specimen 3. Begins fluently but shows marked ataxia toward the end.

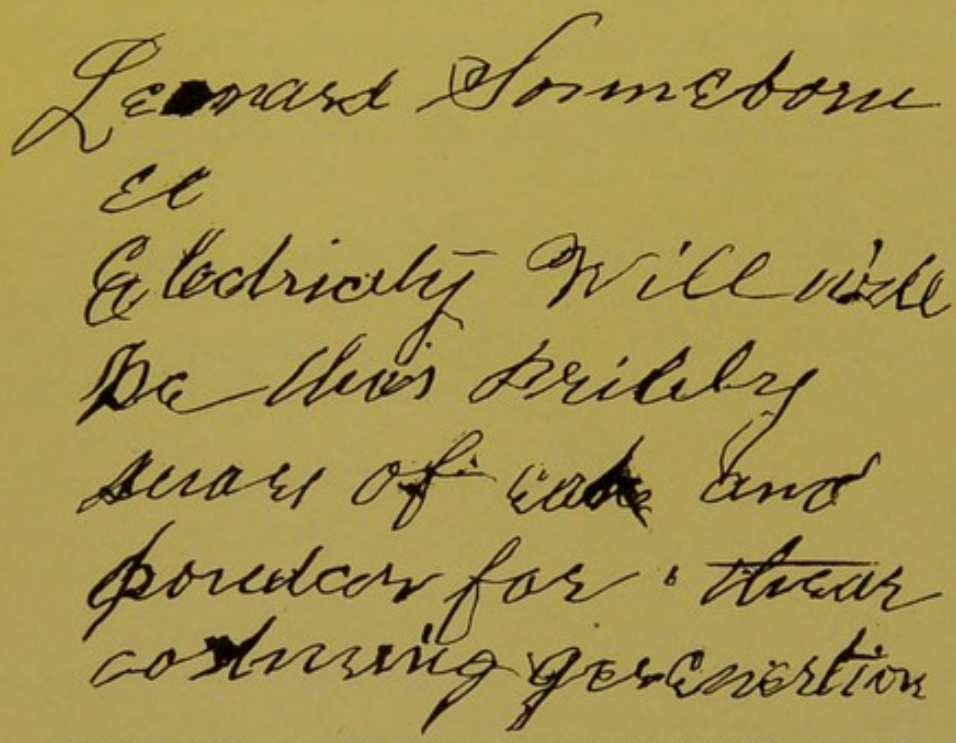
show throughout a wavering uncertainty and there are numerous omissions of syllables and letters; the "i" is reduplicated in "is" and the "F" in "For" is uncrossed. The indiscriminate use of capitals is also noticeable. The introduction of "x" in "electricity" is a very common error in paretic writing. In example 2 the patient attempted to write "I feel very strong and am worth about a million dollars. I will give you a check for 2000 dollars." Besides the laboriousness of the writing, the many omissions and reduplications, one notices the word "other" introduced, which is an instance of perseveration, the letters being a re-arrangement of those in the preceding word "worth." The third example is



Writing specimen 4. Tremor and ataxia.

the spontaneous writing of a very florid patient. Although it shows an apparent fluency, it will be detected that, especially toward the last, the ataxia has made it necessary for almost each individual letter to be constructed separately with a removal of the pen from the paper after each, to regain steadiness. There are also the doubling of the "f" instead of the "s" in "Professor" and omission of "c" in "exactly." Extreme tremor as well as some ataxia are prominent in example 4. Examples 5 and 6 show marked ataxia and in 5 transpositions of letters in "suorc" for "source," "gerenertion" for "generation" occur. In example 7 the "n" in "own" and the "w" in "world" have extra loops. The first "everything" was elided to "ing" and in the

next "th" was omitted. Example 8 is remarkably free from evidence of ataxia or tremor, although the psychosis was quite typical, as shown in the elated content. The awkward attempt to introduce an "l" which was at first omitted in "world" is the only significant point. In example 9 we have an instance of paragraphic writing. The patient had no difficulty in expressing himself orally but was unable to write more than the first word or two of a sentence when it became a senseless jumble of words and letters with no apparent reference to the subject. He would

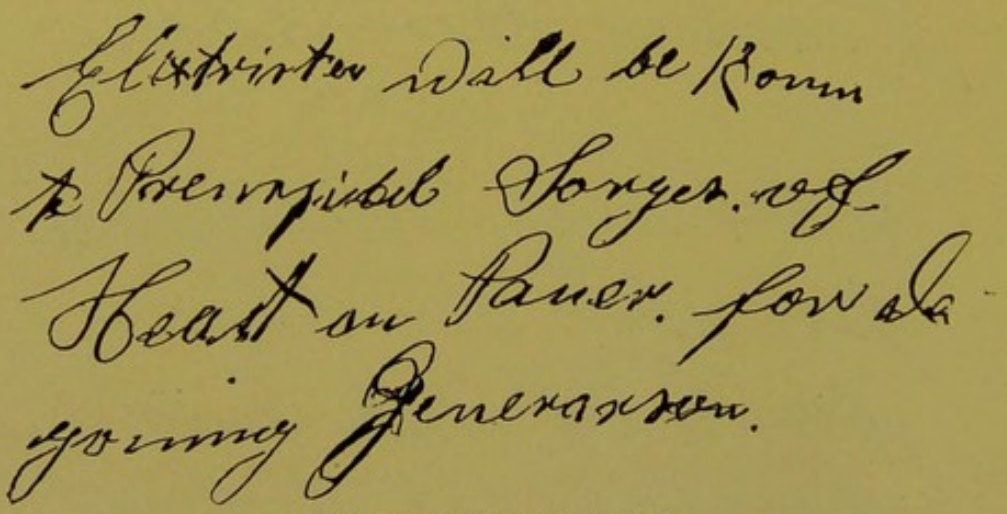


Writing specimen 5. Marked ataxia; transposition of letters and syllables.

repeat constantly to himself a word which had been given him to write but his pen would form something quite different. Perseveration is also quite evident.

Slight attention is paid to the spacing of the writing; the patient does not concern himself whether he runs off the line or the page; the lines run crosswise and obliquely, over and through

each other. He even writes on both sides of the envelope and to different persons on the same sheet of paper. Blots, grease-spots and smudges occur from time to time and often render it impossible to decipher the writing. In far advanced cases there is usually complete agraphia. The patient sits helplessly before the sheet of paper without doing more than to laboriously draw a few wavering lines. Finally, after many futile attempts, he abandons the task, because he has "rheumatism in the hand" or "has no glasses." Examples 10 and 11 show this severest form of writing disorder. In the first, although there is great disorder-

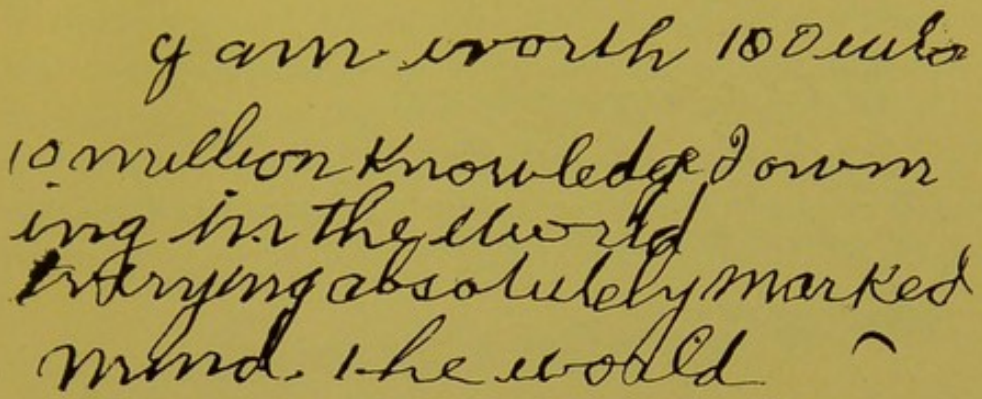


Writing specimen 6. Ataxia.

liness, one can distinguish semblances of letters, while the second is only a senseless scrawl.

The finer testing of the paretic writing by means of the writing balance showed first a certain dragging, but it indicated also that we must distinguish two different forms of disorder in the writing movements, of which now one, now the other is more prominent. As in alcohol intoxication, we observe a loss of the usual intimate relationship between the pressure of the writing on the one hand and the speed and correctness of the characters on the other. In normal writing every alteration in the rapidity

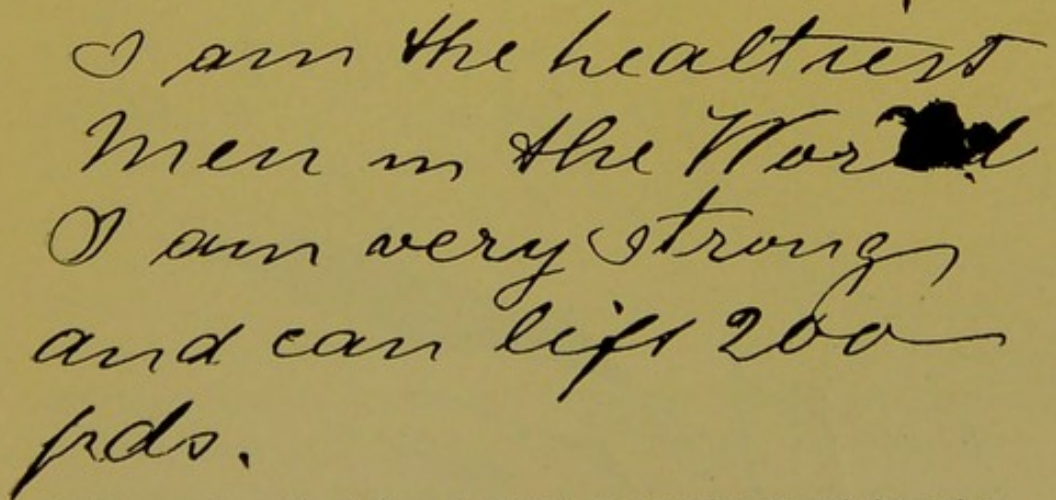
or accuracy causes a corresponding change in the pressure on the paper. The pressure lines of a given specimen afford, to one who can read them, a clear picture of the writing characteristics, and they recur constantly and with striking similarity in the same



Writing specimen 7. Repetitions and omissions; some ataxia.

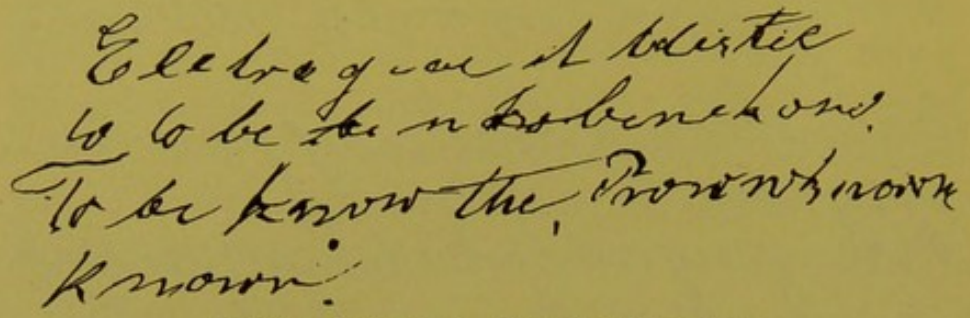
person, because the writing in any one sample gives a type of the manner in which the movements are executed in all the others. Further, in continued writing, under the influence of effort and stimulation, corresponding alterations in rapidity, size, and pressure occur.

All these rules are set aside in paresis; the conformity of the pressure to the different parts of the writing movements is only



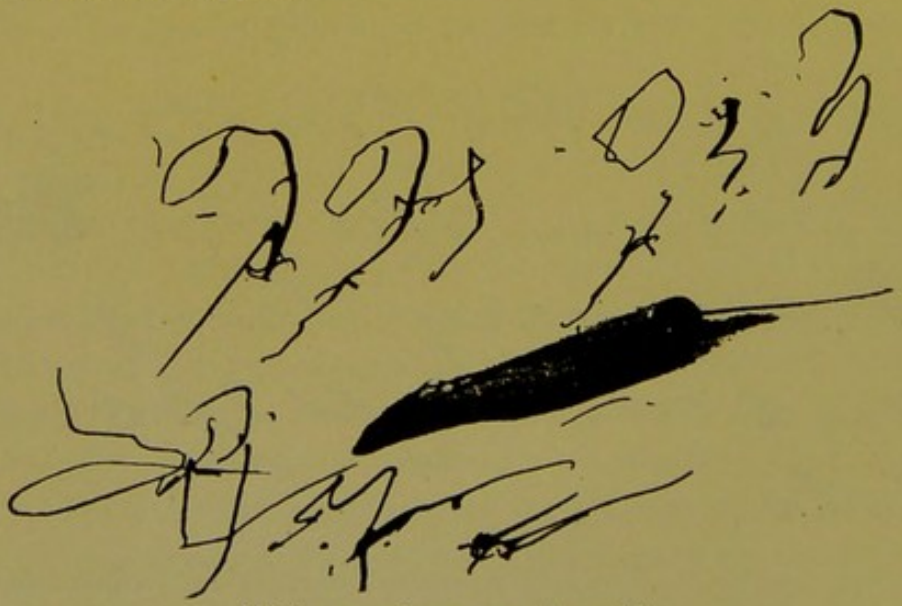
Writing specimen 8. Awkward attempt at correcting an omission, otherwise unusually good.

incomplete, so that the customary variations in the pressure lines are indistinct and irregularly formed; the individual peculiarities of the writing are altered quite independently of each other. On the other hand, many pressure alterations are present which bear



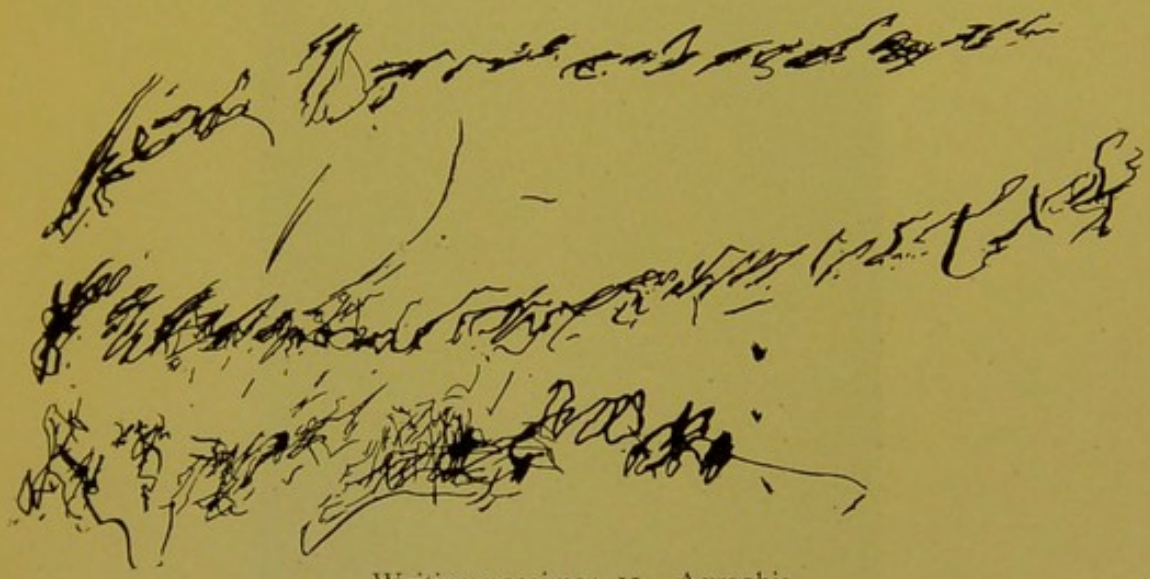
Writing specimen 9. Paragraphic writing.

no relation to the form of the script and which disturb the smooth and orderly course of the writing movements. They cause the already indistinct pressure tracings of the letters to be distorted by unreckoned-with factors. Thus it is that not only is their relation to the individuality of the particular type obscured but the constancy of the pressure lines for the same letter tends more and more to disappear.



Writing specimen 10. Agraphia.

In the early stages of the disease the writing, as a rule, appears to be smaller and the pressure lighter, while the rapidity is lessened,—an indication of the insidiously approaching weakness which endeavors to equalize, to a certain extent, the difficulty in writing by making it smaller; later, with ever increasing slowness, the pressure and size again increase. We can perhaps see in this an indication of the beginning ataxia. The uncertainty of the movements necessitates an enlargement of the writing and more exertion to give the hand steadiness. One must admit that these results correspond satisfactorily with the gross visible changes in



Writing specimen 11. Agraphia.

the paretic writing. They explain, on the one hand, the lack of decision in the writing, and on the other, its irregularity and loss of personal characteristic.

Less pronounced than the disturbance in the fine and complicated functions of writing and speaking are those disordered manifestations of the coarser *movements of the eye*. Of course the paretic very soon becomes incapacitated for all occupations involving especial dexterity, drawing, painting, violin playing, handicraft. I have a small collection of the handiwork of paretic women. In these specimens the disorderliness of the work is notable, the large stitches, the gross technical errors, the loops

dropped from the knitting-needle. At the same time, the mental weakness is evidenced in the fantastic shapes, especially of the stockings; one patient knitted a constricted end the size of a

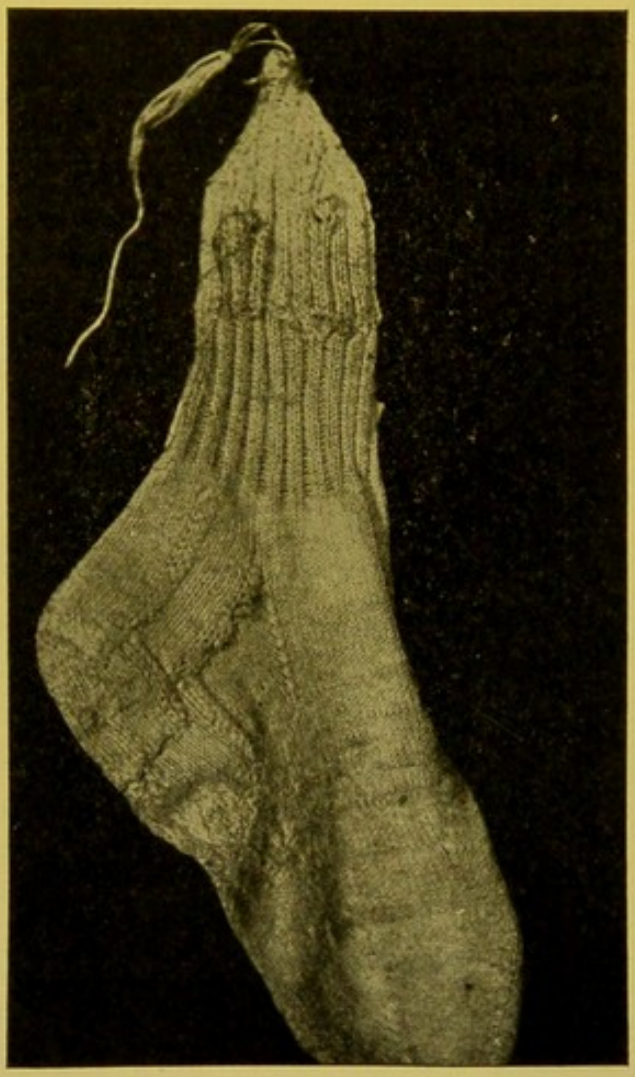


Fig. 2. Stocking Made by a Paretic.

finger onto the already shapeless foot; another made an endless tube; a third closed the stocking at both ends (Fig. 2).

The mechanism of the eye-movements[4] suffers regularly from severe disturbances. Weakness, especially transitory, of the indi-

[4] Rodiet, Dubois, Pamsier, Archives de neurologie, XXII, 128; Mignot, Schrameck, Parrot, l'Encéphale, 1907, II, 6; Raviart, Privat de Fortuné, Lorthisis, Revue de médecine, 1906, 10.

vidual eye-muscles is not infrequent,—in 8.9 per cent. (Junius and Arndt) to 18.2 per cent. (Räcke) of the cases. Complete ophthalmoplegia, however, is exceptional, and then usually in the tabetic form. Now and then one observes restlessness or continuous irregular movements of the eye-ball which render a direct examination almost impossible; nystagmus is frequently present.

The width of the *pupil* shows great variations. Very narrow pupils are relatively frequent, sometimes of pin-head size; mydriasis is less common and generally only on one side. Difference in size of the pupils is observed in 50–60 per cent., according to Räcke even in 83 per cent., but this is also common in other diseases. Changes in the pupil-inequality can occur when there is unilateral loss or weakness of reaction to light, accommodation or lid-closure, but can also be brought about artificially, as Pilcz has shown, since the mobile pupil is now narrower, again wider than the immobile, according to the circumstances. Variations in the pupil width often occur in regular fluctuations (hippus), often one-sided, and in conjunction with nystagmus.

The motility disturbance of the pupils shows a very close coincidence with the establishment of the paretic disease process. Indeed, simple observation shows very frequently (in 74 per cent. of the cases, according to Joffroy) a varying or continuous distortion of one or both pupil outlines, as the first symptom of disordered innervation. The opening is not in the center or is elliptical or pear-shaped; the outline at some place is angular or flattened. These disturbances are important signs of circumscribed paralyses; congenital malformations, residuals of injuries and adhesions must of course be excluded. By far the most important disorder is, however, the failure to contract on exposure to light, the reflex pupillary rigidity, which precedes the usual sluggishness of reaction by some time. Complete loss of light reaction may, on very careful examination, be present in about a third of the cases (Weiler), but for ordinary observation it is

higher (Westphal 50 per cent, Räcke 58.2 per cent., Junius and Arndt 65.8 per cent., Siemerling 68 per cent.). Torkel observed an increase in frequency of pupillary rigidity from 33 per cent. to 41 per cent. during the course of hospital residence. To these are to be added in 30–40 per cent., more or less definite slowness or reduced range of the reaction, the definition of which is uncertain without actual measurements. All these disturbances can be on one side or to a different degree on the two sides. It is not at all uncommon to elicit different results in repeated examinations; perhaps incomplete, transitory, spasmodic states or reflex inhibitions play a rôle in this. I know some cases, however, in which pupillary rigidity after years' standing and frequent confirmation by various observers, has again disappeared; it is, nevertheless, not possible to say with certainty whether these were cases of paresis or of cerebral lues. Westphal has shown that rigid pupils may contract when the eyes are closed very tightly.

With the loss of light reaction, or even before it, the dilatation to pain stimulus also disappears, according to Hirschl's observations; further, that constant, slight variation in pupil-size which one sees regularly in healthy pupils with the aid of a lens as indication of psychic reaction, seems to become less. Weiler has found that in almost all paretics with preserved light reflex, but only in these, the reaction called by him "secondary-reaction" (the consensual narrowing of an already lighted pupil, when the other pupil is also uncovered) fails. Toulouse and Vurpas found a delayed occurrence and prolonged duration of the pupil alteration caused by atropine or eserine. In a large number of cases (according to Joffroy in 22 per cent.) the loss of light reaction may be accompanied by failure to contract on viewing near objects, so that complete pupillary rigidity is present. However, it does not seem, as one might at first think, that this indicates simple exaggeration of the disease process; perhaps we are dealing with a different localization. Now and then loss of accommodative reaction without that of light is observed.

As the most important of all the motor disorders we have finally to speak of the "*paralytic attacks.*" Most frequently they bear the character of cortical epilepsy, less often, that of an ordinary epileptic attack or severe general convulsion of the whole body. There are a variety of prodromal disturbances, unconsciousness, increased dulness, clumsiness in movements, or leaning far over to one side, until the patient suddenly sinks to the floor and the convulsion begins. This consists usually in simple rhythmic twitchings, sometimes also in jerky, throwing movements. One can frequently follow the spread of the irritation step by step to the various parts of the motor cortex. Thus, at first a faint twitching in the facial muscles with rolling of the

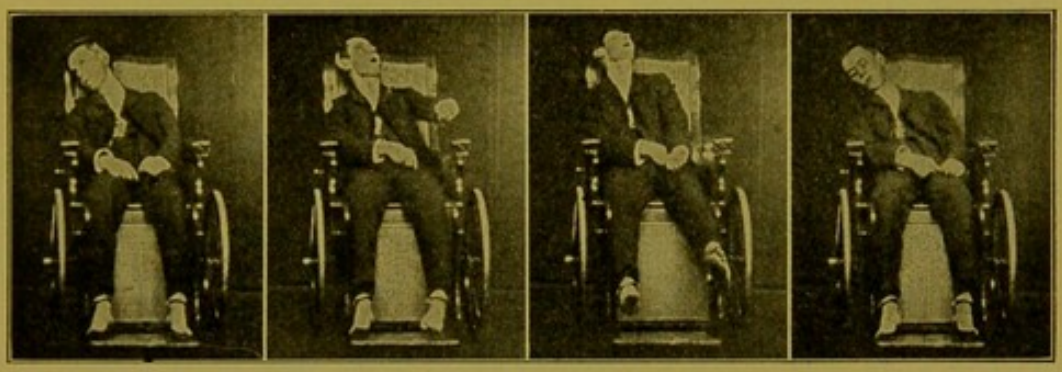


FIG. 3 a, b c, d. Paretic Convulsion.

eyes and nystactic movements occur; then the agitation spreads to the neck, the arm, the respiratory muscles, abdomen and leg of the same side, to seize finally the other half of the body also, while the side first affected perhaps becomes again quiet.

The four pictures, Fig. 3 *a, b, c* and *d,* are taken from a cinematogram and show different stages of an attack. In the first, as the patient sits in a wheel-chair, there is a slight tension beginning in the arms and legs. In the second the head has been drawn suddenly to the left; the fingers are turned in, the arm elevated, the leg is stiff, the feet are drawn inward. The third picture shows the head, which is going through shaking movements, bent backward, the arms stiffened with clenched fingers;

the legs are further extended. In the fourth picture the convulsion has come to an end and the patient has again relaxed and sunk down on the right side. Kemmler has observed that the spasmodic twitchings often show a noticeable synchronism with the pulse-beat and thus seem to be produced by the blood-wave, but Fischer was able to show by tracings that the coincidence in time of the two movements is not a strong one and especially that the twitchings are more irregular than the heart-beat.

The distribution of the convulsion is very diverse. Sometimes only circumscribed areas are involved continuously or with slight variations, such as an arm or one side of the face; in other cases the convulsive movements wander repeatedly through a whole series of muscle-groups. Such attacks may be repeated frequently and successively, sometimes 20 or 30, even 80 or 100 times in the course of 24 hours, with shorter or longer intervals in which the patient lies in a severe stupor or unconsciousness with arms and legs moving, so that a "status paralyticus" exists. Although as a rule the attack terminates after a few hours, still it is not so infrequent that the duration is much longer, even to 14 days. In one patient, I saw, during increasing stupor, twitchings in the right face, then the whole right side, with spastic weakness, hemianopsia and hemianesthesia. Gradually the disturbance seized the left side; there occurred alternating spasms of various muscle groups, complete aphasia, word-deafness and finally death after 15 days.

The body-temperature is usually elevated during an attack, sometimes considerably. In Fig. 4 the temperature chart during two successive paralytic attacks is reproduced. The darkened stripes indicate the night hours. One sees the sudden elevation and gradual falling off of the curves to subnormal; it appears almost as if the second attack gradually prepared itself. The urine often contains albumin. The bowels and bladder are often paralyzed so that retention of urine and fecal impaction may occur with their sequelae of pyelitis, nephritis, periproctitis, if

both functions are not cared for in proper time. Spontaneous taking of nourishment is impossible on account of the paralysis of the muscles of deglutition. Furthermore, since the reflex action of the epiglottis is often entirely lost, there is great danger to the patient from the aspiration of saliva, the mouth cavity containing as it does many decomposition products (occasionally parotitis). In fact we find aspiration pneumonia (the so-called "hypostatic pneumonia") to be the cause of death in the majority of paretics who succumb during convulsive attacks. Finally, with insufficient care, the easily produced pressure gangrene always claims numerous victims.

Recovery from the attack is always gradual, often through a state of unconsciousness or stupor. Not infrequently it terminates in states of confused excitement with impulsive cries. Further, one almost always detects a decided increase in the mental weakness, in some cases marked confusion or even sudden profound dementia after hitherto nearly normal mentality. At the same time there are apt to be all kinds of focal symptoms as residuals, circumscribed or one-sided paralysis, spasms, circumscribed rhythmic twitchings, impulsive movements, speech disturbances, aphasia, word-deafness, hemianopsia, sensory disturbances, which generally disappear, but frequently remain permanently.

A further, rather rare form of paroxysm in paresis is the apoplectic attack which like the ordinary shock comes with sudden unconsciousness, collapse, stertorous breathing, tonic rigidity or flaccid paralysis. It is followed at times by hemiplegia, contractures, aphasic disorders, again there are no residuals. Quite frequently death occurs unexpectedly. Many of the suddenly fatal "shocks" of the middle ages are probably attributable to beginning paresis, as can be shown by the brain-findings in some cases.

Besides these attacks accompanied by severe clouding of consciousness, one sees a number of other disturbances of more or less sudden onset which we may regard as incomplete paroxysms.

The variety of these forms, which are characterized by sudden onset and usually quick and complete disappearance of cortical destructive or irritative phenomena, is very great. To them belong the simple fainting and dizzy spells, with or without the additional transitory difficulty in speech or localized paralysis. Very frequently there is only a slight impairment of consciousness without falling. The patient suddenly becomes very pale or livid. He remains standing but stares, rolls the eyes, does not reply when spoken to; an arm is held stiff or begins to twitch, or the

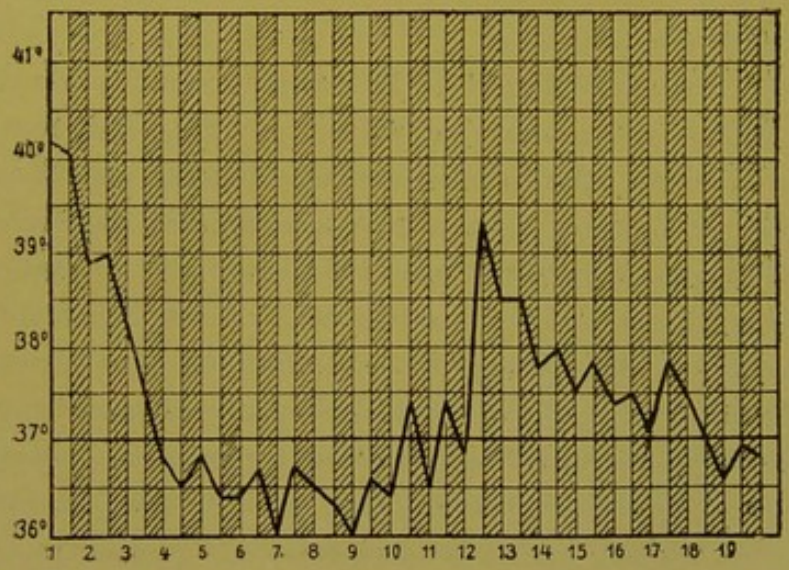


FIG. 4. Temperature curve during two prolonged paralytic attacks.

mouth is drawn to one side; the lips are drawn back from the teeth and after a few moments all has passed. The speech disorders play a great rôle in these spells. The patient suddenly begins to stammer; he does not speak for a whole day or he continually mis-speaks himself, cannot find the right word, calls objects by wrong names.

Sometimes the consciousness remains perfectly clear during an attack. He suddenly loses the use of a hand so that the spoon is let fall, the needle cannot be held or the writing continued; or the hand is spasmodically closed; he kicks out with one leg, falls from his bicycle or from the street-car; the arm trembles; the

speech is thick. Or the patient shakes his head for hours, snaps his teeth together or grinds them; rhythmic twitchings occur in one arm, less often in the leg or one side of the face. Hysterical attacks also are observed now and then but only in patients who have shown hysterical manifestations previously.

On the *sensory* side, there are similar attacks, transitory paresthesias, disturbances of sensation, defects in the visual fields; a hand "goes to sleep"; the fingers get numb; one side seems to the patient to be swollen; he feels as though he were two persons; he has a feeling as if there were fur on the lips; an arm becomes dead and useless for a half a day. Often speech disturbance is coincident with sensory or motor manifestations; the patient feels a cramp in the hand and at the same time cannot speak; the fingers feel as though covered with hair and he begins to stammer. Spells of vomiting are very common and often precede the paresis by years. In many such cases we are dealing with gastric crises, which occur preferably in association with definite tabes but also occasionally with simple posterior-cord paralysis. Similar to these there occur also paroxysmal pains in the back and abdomen with profuse perspiring. More frequently we see episodes of migraine which are shown in unilateral headache and flittering scotoma and which often constitute the introductory symptom of paresis. In other cases there occur temporary fulness in the head, sudden elevation of temperature, difficulty in swallowing or breathing, pains in the heart, some of which at least we may regard as equivalents of paralytic attacks. Neisser conceives the idea of differences in localization of the disturbance and speaks of bulbar, spinal and cerebellar attacks.

In addition to these, finally, we have true *psychic attacks*, quickly recovered-from confusion and loss of consciousness with queer behavior, fragmentary, senseless, irrelevant talking, states of anxiety, sudden impulsive cries or roars, often followed by slight paralytic symptoms or speech disturbance. "He often loses his senses" said the relatives of one of my patients. The

clinical conformity of such disorders with the previously described attacks is so great that, especially in the light of our knowledge of epilepsy, we have a right to speak of them as abortive attacks. All these forms can occur at any stage of the disease, although the apoplectiform attacks belong preferably to the earlier periods. Moreover one observes the lighter spells more often in the beginning, the severer more at a later time. It is not seldom that a paralytic stroke is one of the first symptoms of the approaching disease.

The frequency of the attacks[5] has been given by Obersteiner as 90 per cent., by Heilbronner, from his experience in Munich, as about 60 per cent., by Junius and Arndt in Dalldorf as 53.3 per cent.; Räcke, in Tübingen, saw them in only 34.5 per cent. of his cases. I found them in Heidelberg in about 30–40 per cent. and was inclined to the opinion, shared also by Kemmler, that the bed treatment tended to reduce the number of attacks. Again, in Munich, paretic attacks have been determined in 65 per cent. of the cases before their commitment. Schwanecke was able to prove attacks in 76 per cent. of the Polyclinic patients, mostly spells of dizziness, with or without persisting focal symptoms, more rarely, weaknesses or sudden palsies. Attacks seem to be somewhat more frequent in men than in women; Hoppe found 43.1 per cent. in the former, and only 29.3 per cent. in the latter; Behr found 58.3 per cent. to 51.3 per cent. As immediate causes of the spells may be mentioned excitement, excesses, overloading of the stomach, bowel impaction (colon infection). As a rule, however, a definite precipitating cause cannot be discovered. Their anatomical basis is almost never a hemorrhage or vessel blocking, but only microscopical changes in the cortex; this explains the usually rapid readjustment of the clinical disturbances.

Alterations in the *reflexes* are usually a prominent symptom in paresis. Their behavior, it seems, depends in large part upon

[5] Heilbronner, Allgem. Zeitschr. f. Psychiatrie, LI, 22; Behr, ebenda, LVII, 719; Hoppe, ebenda, LVIII, 1079; Räcke, Arch. f. Psychiatrie, XXXV, 547.

the location of the spinal cord changes which are always present. These involve rather seldom the lateral columns alone, according to Fürstner 14 per cent., from Torkel's[6] observations 12.6 per cent. of the cases, somewhat more frequently the posterior columns alone, but most often both together. In the spastic form of the disease there is an increase in the general reflex excitability, sometimes so great that a strong thrust toward the patient's face will cause a drawing together of the whole body. The testing of the reflexes proves to be very difficult in these cases, since the patient cannot relax his muscles completely. When one finally succeeds by means of the known artifices (mental diversion, Jendrassik's method) he finds the reflexes, in about two thirds of the cases, more or less greatly increased, so that ankle-clonus and sometimes reflex spasm (on placing the toes on the floor) occur. Not infrequently one sees a disappearance of reflexes which were at first exaggerated, as well as many variations in their activity. In 18 per cent. there is a difference between the two sides (Räcke).

In a small group of cases which is reckoned by Junius and Arndt as 29.6 per cent., Fürstner as 24 per cent., Torkel as 16 per cent. and Schwanecke as 19 per cent., we have to deal with more or less uncomplicated disease of the posterior columns. Here we find diminution or loss of the patellar reflex, ataxia, Romberg's sign, bladder and intestinal paralysis, less commonly hypotonia, girdle-sensations, lancinating pains and crises. In these cases there is especially apt to be reflex pupillary rigidity and miosis. Distinct tabes develops according to Torkel in 6 per cent., according to Hirschl in 8.5 per cent. As a rule it precedes by a long time the outbreak of paresis, often over a decade (ascending or tabes-paresis). In two thirds to three fourths of

[6] Gaupp, Über die spinalen Symptome der progressiven Paralyse, 1898; Torkel, Besteht eine gesetzmässige Verschiedenheit in Verlaufsart und Dauer der progressiven Paralyse nach dem Charakter der begleitenden Rückenmarksaffektion? Diss. Marburg, 1903.

the cases the spastic and the posterior-column symptoms are intermingled in the most diverse manner. Repeatedly a return of lost knee-jerks is observed after a paralytic attack even on the uninvolved side (Westphal).

The other tendon reflexes, especially the Achilles, agree in general, but not always with the knee-jerks. The cremaster and jaw reflex also show similar alterations according to Marandon de

Fig. 5. Paretic Contractures.

Montyel. Babinski's reflex is frequently found accompanying prominent spastic symptoms; it is fairly common, usually on one side, after an attack. In much demented patients one sometimes observes the reflex described by Dobrschansky, the making of sucking movements when the lips are rubbed, in reality an instinctive act.

As the disease progresses, the patient becomes continuously bed-ridden, since he is unable to walk, to stand or even to sit up.

It is the rule for fixed contractures to form, first in the legs, then in the arms; the adductors of the thigh are also apt to be rigidly contracted. The result is shown in Fig. 5. The arms are flexed, the fingers clinched, the thighs crossed over each other. The patient's entire body is stiff and immovable and can only be lifted or rolled as a whole like a bundle, without change of position of the different members. The musculature shows a general, sometimes severe, reduction which, however, is not accompanied by reaction of degeneration but only by a reduction of electrical excitability. In these last stages of the disease one can often observe separate muscle-groups which are spasmodically tense and show more or less violent twitchings. This is especially noticeable during active or passive movements but is seen also during rest. Once I saw a crossed radialis and peroneus paralysis, without doubt of neuritic origin; Möli has described a number of similar cases. With these belong the rare, isolated muscular atrophies. Some cases are described in which syringomyelia was present, others with spinal muscular atrophy or amyotrophic lateral sclerosis; association with multiple sclerosis has been observed. Often a striking symptom, even in the early stages, is the stooping position of the head, which does not rest upon its base but is constantly supported by the stiffly contracted neck-muscles. Petrozzini mentions that such an attitude could not be held by a healthy man longer than ¼ to ½ hour, and thinks the symptom is therefore diagnostically important.

Of late years, the findings in the *cerebro-spinal fluid*[7] have assumed a position of clinical importance.

First the experiments of Schäfer[8] have thrown some light on the *pressure* within the spinal canal. He found in 25 paretics, an average pressure of 182 mm. when lying on the side; in two thirds of the cases it was between 150 and 380 mm. This, com-

[7] Clergier, La ponction lombaire chez les paralytiques généraux, Thèse, 1905.

[8] Schäfer, Allgem. Zeitschr. f. Psychiatrie, LIX, 84.

pared with the normal 40–70 mm., is a very marked increase; in two cases the pressure sank shortly after attacks, from 200 and 270 mm. respectively, to 60 and 80 mm.

Schaefer found the *albumin content* of the fluid considerably increased; it varied between 0.75 and 3.5 per cent. with an average of 1.23 per cent., against the normal of 0.2–0.5 per cent., an observation which has been repeatedly confirmed. According to Guillain and Parant, this is, in part, serin, while in health only globulin is present, which is precipitated by magnesium sulphate.

Nonne and Apelt[9] have described a method for the separate determination of *globulin and nucleo-albumin* (*"Phase I"*), to which much importance is ascribed. A saturated ammonium sulphate solution is added to an equal quantity of spinal fluid and let stand for 3 minutes in the cold; the more or less definite cloudiness which is then noticed indicates the presence of the substances mentioned. All cases of paresis, nearly all of tabes, cerebro-spinal lues, congenital syphilis and brain abscess, as well as a few cases of extra-medullary tumor, give this reaction. In secondary syphilis and in tertiary without nervous involvement, in multiple sclerosis, combined system disease, and a number of infectious diseases, the precipitation occurred much more seldom; in old lues without active manifestations, in epilepsy, alcoholism and other psychoses and neuroses it is absent. Pappenheim has expressed the opinion that the spinal fluid of the paretic destroys leucocytes very quickly; on the other hand it has been shown to be innocuous for rabbits.

Of far greater importance than this and the many other findings (cholin, cholesterin, increased phosphoric acid content, behavior of the freezing point) has proved to be the *cytological examination* of the spinal fluid. The very extensive studies of the last few years have shown conclusively that in paresis, with few exceptions, there is a more or less excessive increase of the cellular element of the spinal fluid, which is quite independent of the

[9] Nonne und Apelt, Arch. f. Psychiatrie, XLVI, 357.

albumin content. It is, at the same time, similarly frequent and marked in tabes, syphilis of the nervous system and hereditary syphilis, less common and not so definite in secondary and latent syphilis. The same is true of brain tumors, multiple sclerosis, apoplexy and for occasional cases of other forms of disease,

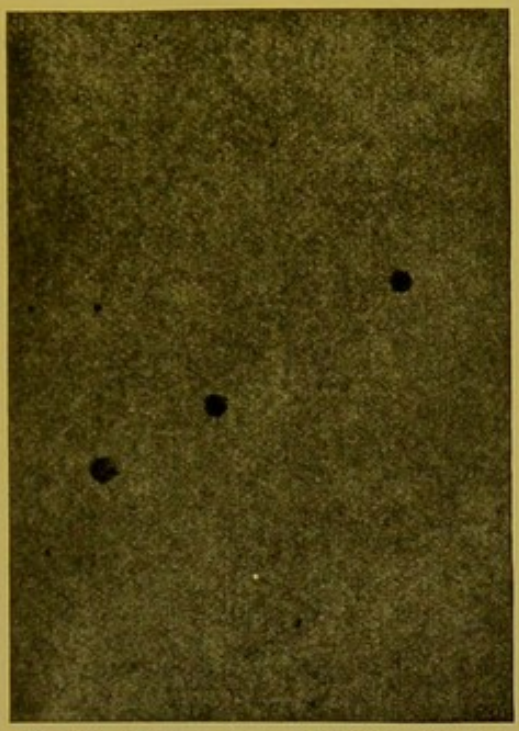


FIG. 6. Cells in Spinal Fluid in Health.

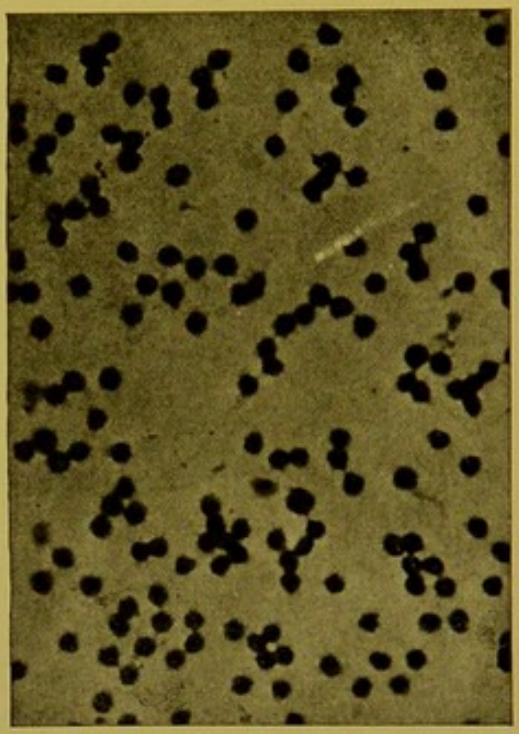


FIG. 7. Cells in Spinal Fluid in Paresis.

obviously with the possibility of the existence of previous lues. In paresis, the lymphocytosis which in the first place only points to a syphilitic origin may be present in the beginning of the disease, possibly long before; it may disappear in good remissions and, perhaps, just before death. It is reduced by anti-syphilitic treatment; it seems also to temporarily disappear under other circumstances, since occasionally in the same patient a cell increase is present at one time and not at another. Cases with repeatedly normal findings are so rare that the correctness of the diagnosis may be justly doubted.

Figs. 6 and 7 will give an idea of the cytological findings in paresis; the centrifugate from a normal and from a paretic spinal

fluid are shown side by side. According to Nissl's method 4 to 5 c.c. of the cerebrospinal fluid is drawn into a narrow pointed glass tube and revolved for 30 minutes in a centrifuge, the fluid is poured off very carefully, the precipitate taken up in a very fine capillary pipette and deposited on a cover-glass; the drop should not have a greater diameter than 5 mm. The preparation is fixed in alcohol-ether and stained with polychrome methylene blue. One notices in the illustration that in the same area the normal spinal fluid contains only 3 cells while the paretic contains a large number. By means of the cell-chamber of Fuchs and Rosenthal, the number of cells per cubic millimeter of cerebrospinal fluid can be estimated; they number 8–10 at most in health but in paresis may reach several hundred.

Alzheimer has succeeded in determining the histological nature of the cells in the spinal fluid. The lymphocytes, which are the only cells found in normal spinal fluid, are greatly increased in numbers in the paretic fluid and show numerous atypical shapes. Plasma cells, the significance of which in the paretic process will be discussed later, are also numerous. Macrophages, with ingested cells and degeneration products, are found and kinetic figures are occasionally seen. Not only the number, but also the variety of the cells is much greater; the occurrence of plasma cells and phagocytes must be mentioned as especially characteristic. Widal and Lemiere as well as Hartman mention the occurrence of leucocytes in connection with the paretic attacks, but one must here consider an infectious origin.

An unexpected advancement of our knowledge of the nature of the paretic process has been furnished by the *serological studies of the spinal fluid*. Plaut[10] has shown that the Wassermann reaction, the deviation of complement by addition of watery ex-

[10] Wassermann-Plaut, Deutsche med. Wochenschr., 1906, 44; Plaut, Die Wassermannsche Serodiagnostik der Syphilis in ihrer Anwendung auf die Psychiatrie. Habilitationsschrift, 1909, translated as No. 5 of this series by Jelliffe and Casamajor; Marie et Levaditi, Bulletin de la société de méd. ment. de Belge, 1907; Kafka, Monatsschr. f. Psychiatrie, XXIV, 529; Stertz, Allgem. Zeitsschr. f. Psychiatrie, LXV, 565.

tract of congenitally syphilitic liver to the spinal fluid of a paretic, occurred in about 95 per cent., while it was found, even in tabes and cerebrospinal lues, only in a small number of cases. In cases with a very rapid course the reaction appears to be especially strong, while in those with long duration and remission it may be quite weak. The Wassermann reaction is independent of the lymphocytosis. This has been confirmed many times. Inconsistent results may be attributed in most cases as Plaut has contended in detail, to changes in the very difficult technique, sometimes also to disturbance of the alkalinity of the blood.

On the part of the *bladder* there are frequent disturbances which are independent of paretic attacks; they consist of sphincter paralysis as well as retention, the first more commonly than the latter (urine dribbling).

The *abdomen* is frequently subject to tympanitic distention, apparently resulting from atony of the intestinal musculature. The sluggishness of the bowel may lead to excessive impaction. On the other hand, in all advanced cases there is a complete inability to retain the feces, perhaps due partly to paralysis of the sphincters, but chiefly because the patient is not aware of the approaching evacuation nor the distention of the bladder even to the umbilicus.

The *sexual strength* is lost, sometimes after a period of increased sexual activity. Marandon de Montyel found in 79 per cent. of his cases a complete loss, in 15 per cent. an increase of sexual power.

Among the *vaso-motor disturbances* are to be mentioned in the first place the frequent fulness in the head, erythema, long-continued redness of the skin and even the formation of wheals with slight irritation, transitory edema, cyanosis. Occasionally there is a tendency to extensive ecchymosis upon slight trauma. The sphygmograph curve often shows a gradual rise and a reduction of the summit-wave ("retarded" pulse), signs which point to a prolonged and weakened expansion of the vessel wall. The

palpable arteries, particularly the temporals, are often very tortuous, prominent and rigid from loss of elasticity and thickening of the wall. According to Audenino and Parazzolo the blood pressure is sometimes increased, sometimes diminished, the latter more commonly in the last stages of the disease. It shows marked variations from day to day, often indeed during one sitting; marked differences of variable degree in the two sides are observed. Bodington found the blood-pressure elevated in depressed states and lessened in excitement. Plaskuda observed a slight rise in the pressure preceding a paretic seizure, then a sinking immediately before the convulsion and a marked elevation during its progress followed by a gradual return to normal. The same phenomenon was demonstrated on the following day by Audenino.

There are a large number of symptoms accompanying paresis the occurrence of which one may often regard as the result of degeneration in certain *trophic* nerves governing the nutrition of the organs. To this group belong herpes zoster, pemphigus-like eruptions and the related peeling of the skin, described by Zahn, in circular or irregular areas which do not correspond to definite nerve distributions and are not exposed to pressure. Although he found alterations in the spinal ganglia and the nerves, Zahn is inclined to look for the cause not in these but in some toxic agent circulating in the blood; infectious processes are also to be thought of. In this connection the bed-sores, rib-fractures and swollen ears are also to be mentioned, as well as the frequent pneumonia for which a remission in the vagus-innervation may be responsible. The irrefutable, scientific and even more practical work of von Gudden has afforded the proof that all these disturbances are not from internal causes but develop without exception, under the influence of external injuries.

One can hardly avoid the assumption of a general reduction in the resistance of the tissues in paresis as a contributory cause, since very severe lesions may result from relatively insignificant traumata. The occurrence of bed-sores may be explained in the

first place by the fact that the patient on account of his blunted sensibility does not notice, as would a healthy person, an uncomfortable sense of pressure, and for this reason, or because his helplessness renders it impossible, he does not change his position but lies like a log in bed. One or two hours can, under these circumstances, produce redness, vesicles and even blisters, especially where the legs lie crossed or come in contact with the hard edge of a chair, while a single night without attention suffices to

FIG. 8. Recent Hematoma of Ear.

FIG. 9. Old Hematoma of Ear.

cause a sore several centimeters deep. In addition to this, the uncleanliness and the lessened resistance of the patient naturally furnish favorable soil for the development of infectious skin lesions, such as furuncles. Fractures of ribs and hematoma auris occur frequently in paresis and sometimes to a frightful extent, because the patients are very awkward, at the same time often restless and especially because they do not defend themselves or complain and therefore suffer helplessly the mistreatment of their fellow-patients.

The pictures, Figs. 8 and 9, are from plaster casts of a fresh and of a shrunken hematoma of the ear. In the first there is an enormous globular swelling due to extravasation of blood; in the latter, thickening and disfigurement of the cartilages after absorption of the blood. It is an undoubted fact that, with better organization and supervision of the corps of attendants, the number of broken ribs and contused ears becomes less. Predisposing causes, however, certainly play an important rôle; these include disorders in nutrition of the ear-cartilages and extreme fragility of the ribs, conditions seen frequently enough in the cadaver. The latter appears to depend upon a loss of bony material with

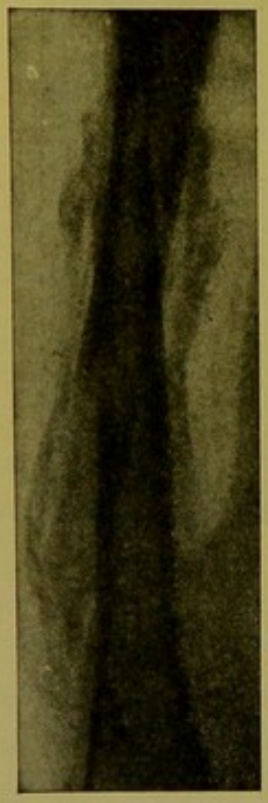


FIG. 10. Spontaneous Fracture of Humerus in Paresis.

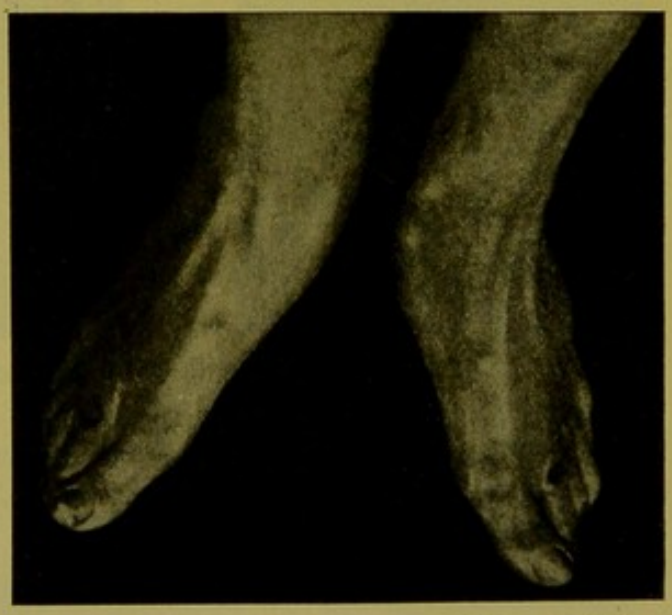


FIG. 11. Bi-lateral Perforating Ulcers in Tabes-Paresis.

substitution of fat and is a part of the general nutritional disturbance in paresis. Certain importance is also attached to the

lessened use of the ribs as a result of the reduction in breath-movements. Marked atrophy occurs also, however, in the other bones, although it does not so often lead to fracture. We observed a spontaneous fracture of the arm whose Roentgen picture is

Fig. 12. Arthropathy in Tabes-Paresis.

shown in Fig. 10, in a seventeen-year old juvenile paretic, simply through the muscular effort during a convulsion. One notices the slender form of the bone, the marked displacement of the fragments and the excessive callous formation. Such occurrences are comparable with the arthropathies and with the perforating ulcers which are occasionally seen in paresis. Fig. 11 is a photograph

of bilateral, symmetrical perforating ulcers of the feet in a woman with tabetic paresis; Fig. 12 is that of an arthropathy of the knee-joint in a patient with the same form of disease.

Disturbances of the bodily *temperature* are extremely common in paresis. The two sides may show marked difference. Temporary but quite decided elevations of temperature are often observed without there being any discoverable cause therefor. Sometimes an enema will reveal an accumulation in the bowel, the bladder is distended or a broken rib is discovered. In other cases slight bronchitis or pneumonic disturbance constitutes the basis. Less often the febrile movement is directly connected with the disease in the brain. Such a relation probably exists in the fever which usually accompanies paralytic attacks, although when the seizure lasts for a long time other causative factors such as aspiration pneumonia come in. In the last stages of paresis there are often prolonged temperature depressions which are borne remarkably well by the patient. An example is shown in Fig. 13

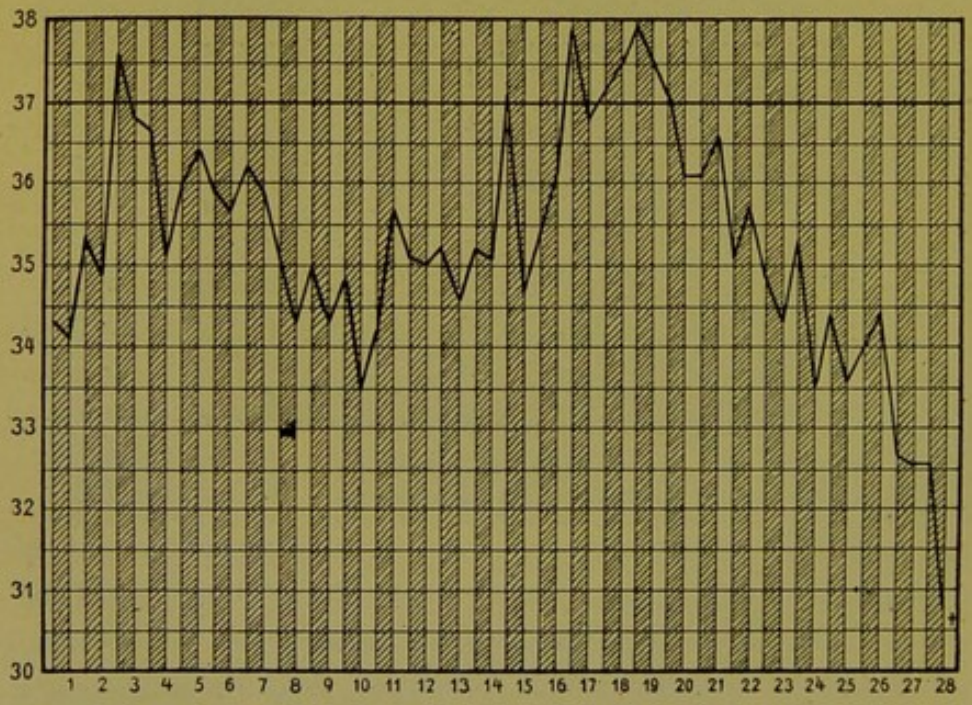


FIG. 13. Terminal state of paresis. Excitement; continuous subnormal temperature.

which gives the course of the temperature during the last four weeks of life in an excited paretic with very expansive ideas. Only a few times was 98.6° F. overreached; the temperature remained generally between 93.2° F. and 95° F., and finally, although still during life, sank to 87.5° F.; several times it rose during the night and dropped in daytime. During this time, with abundant nourishment, the weight sank with fluctuations and by no means rapidly.

Of the other functions of the organism those which are profoundly disordered in paresis are especially the sleep, the appetite

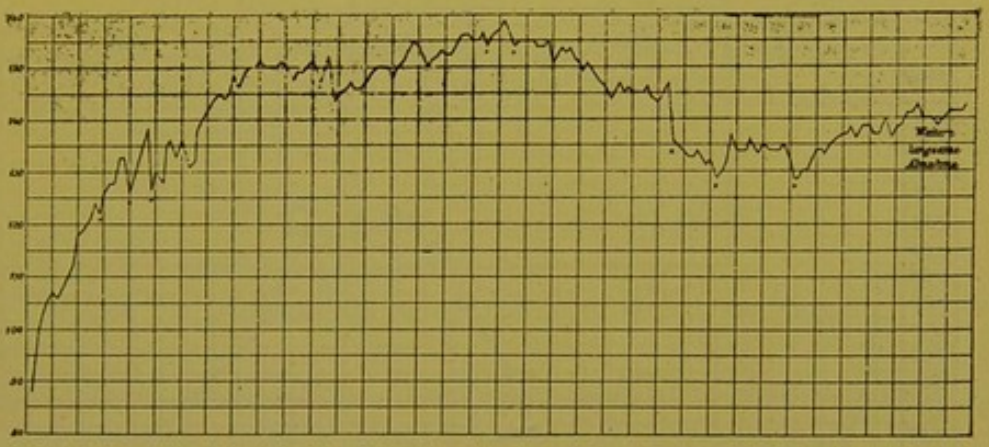


FIG. 14. Demented paresis with initial excitement. Many convulsions (*).

and the weight. The *sleep* is often greatly disturbed in the early stages, later in the excited states frequently abolished for a time, while toward the end it is better, although in the demented torpor of the patient an opinion on this point is hardly possible. In many patients an actual somnolence develops so that they are only occasionally fully awake, when eating or when conversed with, after which they immediately renew their sleep. The *appetite* is usually diminished in the beginning and in excited states, only to become truly ravenous in the later stages. Rumination is sometimes observed. The general state of the body often offers the appearance of a premature senescence, grayness of the hair, loose, wrinkled skin, pale features, weak musculature, bent figure, poor teeth.

The body *weight* falls in the beginning and at the height of the disease, but later, with continuous rest and accumulation of adipose, it increases markedly, until far above the normal; sometimes 7–8 pounds are gained in a week. Finally, at the end it falls irresistibly to a condition of profound marasmus. I saw one patient gain 53 pounds and lose 86. The terminal diminution in weight takes place even though the patient eats greedily and in large quantities. A part of the course of the disease is shown in Fig. 14. It begins with the very low state during the initial excitement; then occurs a very rapid rise, which is only broken from

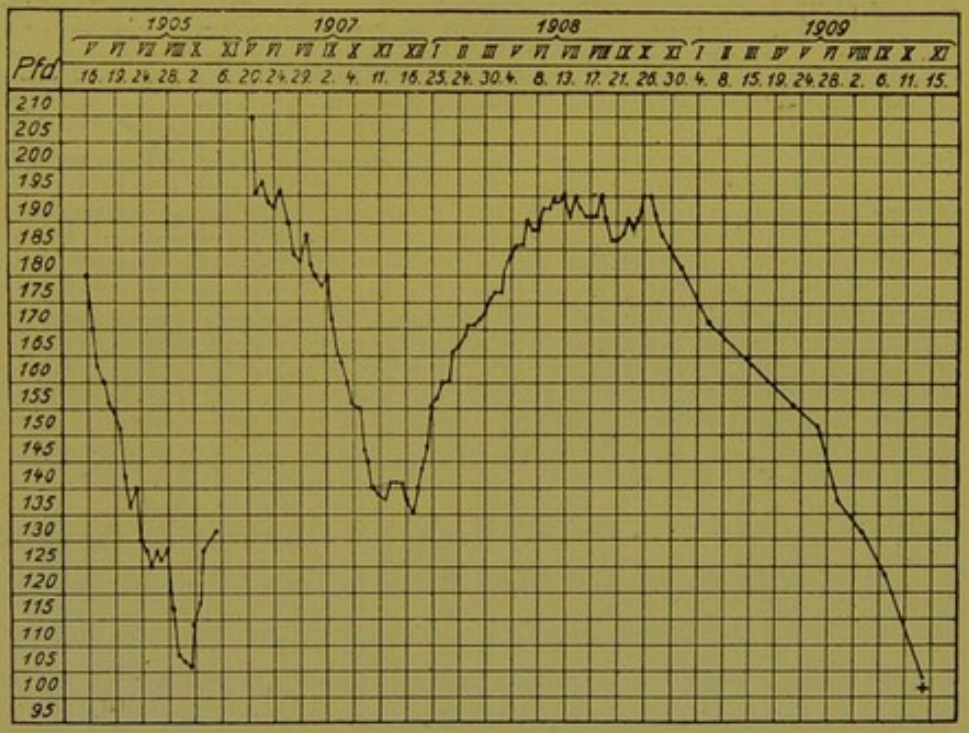


Fig. 15. Excessive variations in the body-weight in paresis.

time to time by a slight falling-off, generally as a result of convulsions which are indicated by small stars. After more than two years a gradual reduction begins which is thereafter progressive. The patient died after 4½ years in the institution.

A much more marked change in weight is shown in Fig. 15. The patient went into a rapidly increasing excitement and quickly

lost 74 pounds, but improved in the clinic and at home to the extent of putting on 104 pounds. In another excitement he again lost 74 pounds. When he quieted down an inordinate appetite developed, from which he again acquired that peculiar fatness of the paretic only to lose it finally in a sudden decline to 90 pounds at the time of his death. Fig. 16 shows the course of the body-weight in a patient with prominent dorsal-cord symptoms who went through an acute excited period with expansive ideas during his first residence in the hospital, then for years was able to

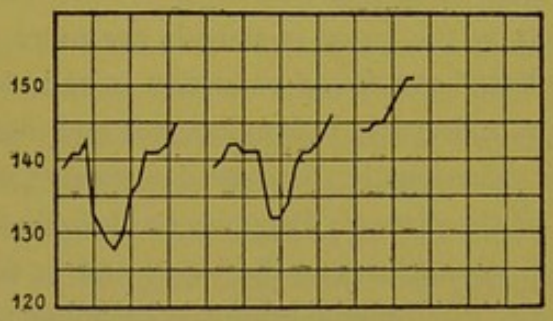


FIG. 16. Body-weight in a paretic with 5-year remission.

be employed as an officer. Five years later after several paretic convulsions he returned to us, this time in a depressed state. The weight, however, showed a variation very similar to that of his first institutional confinement. This time also there soon followed an improvement in the psychic condition with marked increase in weight, but he was re-admitted a few weeks later after a serious attempt at suicide.

The bulk of the disturbances last mentioned go to show that, in paresis, we are dealing with a severe general *disorder of nutrition*. Unfortunately we are as yet able to form only a very incomplete picture of this. Kaufmann[11] who carried out extensive experiments on the metabolism in seven cases reported very variable results, which indicated variations and sudden reversals of relations, great irregularities in the nitrogen and water

[11] Kaufmann, Beiträge zur Pathologie des Stoffwechsels bei Psychosen. I. Die progressive Paralyse, 1900.

balance, transitory disturbances of oxidation with accumulation of intermediate substances, finally acetonuria and glycosuria, incomplete thermic regulation. Dupré and Tissot have found that the elimination of methylene blue is retarded and irregular; Jach ascertained that levulose administered during fasting appeared in the urine in paretics much more often than in other mental diseases or in health. The nitrogen elimination in the urine is sometimes diminished, sometimes increased; often, especially after an attack, albumin and sometimes sugar appear. The phosphates are increased after a convulsion.

The *findings in the blood* in paresis are more consistent. A number of authors have found reduction in red-blood corpuscles and hemoglobin, a decrease in lymphocytes and increase in leucocytes. Klippel occasionally discovered nucleated erythrocytes in the beginning of the disease; Pappenheim describes a paroxysmal increase of the polynuclears in the blood and in the spinal fluid. Bodington[12] saw the "finely granular oxyphile cells" increase toward the end of the disease, while the mononuclear lymphocytes, the "hyaline" and the eosinophile cells showed a reduction. Diefendorf observed in the last stages of paresis increase in the hemoglobin content and in both red and white cells, especially the polynuclears; this may be due to a decrease in the watery element resulting in a gradual concentration of the blood. The blood changes are very pronounced in paretic attacks. Just before the onset there is an increase in hemoglobin and red cells; during the spell Bodington found the white blood cells multiplied sometimes five or six times. Only the finely granular oxyphile cells were concerned, while the mononuclear lymphocytes and eosinophile cells were diminished. These alterations are gradually righted after 2 or 3 weeks. The alkalescence of the blood was found by Bornstein to be slightly reduced, the amount of fibrin sometimes increased, the lecithin-content greater or at the upper limits of normal. Peritz also observed an increased amount of

[12] Bodington, Archives of Neurology, III, 143.

lecithin in the blood, also the excretion of large quantities of lecithin in the stools, while the amount of this substance in the bones was reduced to, at most, one tenth of the normal. Agostini mentions the diminution of the isotonicity of the blood; d'Abundo and Rebizzi noted its toxicity; Benigni found lessened resistance of the red-blood corpuscles to the electric spark. Idelsohn[13] carried out detailed experiments on the bacteriolytic property of the blood. He found this capacity entirely lacking in nearly half his cases, while it was absent in only 4 per cent. of the control cases; in another fourth of the paretics it was much reduced.

Regarding all these observations a doubt must remain for the present as to whether and to what extent they stand in causal relation to the paretic disease-process, but the connection of the *serological findings in the blood* therewith may be regarded as certain. Plaut reported 245 cases of paresis with positive Wassermann reaction in the blood-sera, while only two anatomically confirmed cases gave a negative finding; Edel, also, in 54 paretics did not once fail to obtain complement-fixation in the blood. A certain degree of alkalescence seems to be necessary for its evolution. Since we know that this sign appears with the development of syphilitic symptoms and again disappears, we have all grounds to regard its constant presence in paresis as an indication of the syphilitic origin of that disease.

[13] Idelsohn, Arch. f. Psychiatrie, XXXI, 640.

CHAPTER II

Clinical Forms

The diversity of the disease pictures which our experience constructs out of the several disorders which have been spoken of is so great that even a partially satisfactory survey of the clinical groups of paresis is hardly possible. Although we meet with the common features of the peculiar mental weakness, the signs of an organic brain disease and finally the progressive course leading inevitably to a wiping-out of the mental and physical life, still the observations in their development as well as in their setting may differ so from one another that, to the inexperienced, the homogeneousness is obscured through the influence of conflicting details. Long experience teaches us that all the apparently different forms may change suddenly and unexpectedly from one to the other and that only the previously-mentioned cardinal features form "the fixed point in the flow of the symptoms."

In this multiplicity of disease-pictures, which can hardly be reviewed briefly, certain types of course may be differentiated, but then insurmountable difficulties are encountered. If we distinguish, as is the usual custom, the following principal forms of paresis, the demented, the depressive, the expansive and the agitated, we must not be deluded into believing other than that such a grouping is entirely arbitrary and that its only value is to facilitate the presentation of the subject. The same holds for any other of the numerous attempts to classify the clinical material solely in a basis of psychic or physical signs. Thus Sérieux and Ducoste differentiate expansive (27 per cent.), sensory (24 per cent.), demented (24 per cent.), persecutory (3 per cent.), depressive (2 per cent.), circular (7 per cent.), hypochondriacal

(7 per cent.) and manic (6 per cent.) forms. Junius and Arndt add to the forms mentioned by me a group of mixed (10.42 per cent. men and 9.07 per cent. women) and one of uncertain (0.87 per cent. men, 0.22 per cent. women) cases. Perhaps a better knowledge of the etiological or the pathological-anatomical relationships of the disease will enable us, under a new point of view, to evolve a more reliable grouping. Of the attempts which have already been made in this direction we will speak later.

Demented Form.—The form of paresis which is, at least at present, by far the most frequent and in which the degenerative symptoms are most prominent is the demented form. In it the progressive deterioration with paralysis dominates the picture from the beginning. The occasional irritative symptoms are, in this form, only transitory and indefinite.

The first indications of the approaching disease are loss of mental acuteness, inability to work, poverty of thought, forgetfulness and absent-mindedness, sudden moodiness and irritability, besides an unaccustomed indifference and laxness in important matters, complaints of pains and feeling of pressure in the head. The patient tires quickly, sleeps when in company, sometimes appears suddenly to lose himself; he is uncertain and easily influenced in his opinions and conclusions, but on the other hand may be particularly capricious and obstinate over trivial matters. He makes mistakes in things with which he is entirely familiar and has to ponder a long time in figuring out simple dates with which he has perhaps been working every day. Consciousness gradually becomes clouded; the patient is no longer able to comprehend what occurs about him, does not recognize money, is not clear as to time, place or situation, cannot orient himself correctly. The year 1900 was falsified by the writer, thought one patient. His thoughts are confused; he has sometimes the appearance of a drunken person, mistakes a neighboring street for his own or is confused in his own house and perhaps no longer recognizes his own family and friends.

Fleeting delusional ideas occur not infrequently. The patient sees black men with huge beards, angels in the sky, hears knocking, noises of insects, abusive epithets, people inside of him who give him commands and whom he tries to blow out with his breath, he feels as though doubled or bewitched. He has a heavy sensation in his head, he must be insane, dead, or he has lost his tongue, has no mouth, cannot make water, cannot speak or eat, is ruined by bad flesh, is dried up, says: "Those are not hands?" Someone has accused him of murder, they will hang him; robbers come; his wife is untrue to him. Or he feels absolutely healthy, possesses great weight, is very handsome and very strong, can lift 400 kilos, has begotten a royal prince, his wife has breasts like a fatted cow. He is of the nobility, a general, Charles Augustus, emperor of Berlin, professor of all branches, has 28 names, has "magnificent drawers," a silk cap, gold clocks, 1,000 elephants and many orders, glass-houses filled with gold, three millions in the savings bank. Every afternoon he has a wedding to which all the princes are invited, he will lend a million to America, will have himself divorced so he can marry a rich woman. Women patients have silk dresses, a golden carriage which calls for them, "diamonds and rubies"; they have "had the finest gentlemen," desire to marry another husband, have had a child by the doctor. One patient in the beginning of the disease telegraphed home that he had made a great discovery. Shortly afterward he jumped from a window in an attack of anxiety, subsequent to which he presented a picture of simple, cheerful dementia.

The delusional ideas of the patient bear the stamp of childishness and weak-mindedness; they are usually very easily influenced by persuasion. Often the patient begins to confabulate in the same simple manner, to tell of a meeting with the emperor, of a shipment of gold which should arrive, of a visit which he has made that morning. In some cases the memory disorder, with or without retrospective falsification, is so prominent that a picture

like that of Korsakow's disease is presented. The patients forget in the briefest space of time what has just happened. In spite of retained faculties they have no inkling of their location or surroundings, think themselves in dream-like situations, tell of remarkable experiences.

The excitability of mood usually diminishes steadily. In the beginning there is often a dull anxious state, mental restlessness, praying, whining, monotonous muttering or sudden change of disposition, causeless laughing, senseless grinning. Many times there is excitability and a tendency to violence which shows itself in reckless abuse, blind destructiveness as well as in threats and assaults against those about him. Interspersed with these states are acute delirious excitements, especially at night, in which the patient cries noisily, mistakes his surroundings, gets everything confused, wanders about the house. Many patients show, for long periods, an instinctive restlessness, cannot remain seated, leave the bed at night, run away aimlessly, spend the night in the open air. Later the patients become more dull and apathetic, do not show the slightest interest for persons or things which concern them most intimately. The reproaches which they receive for their errors are taken indifferently; they hardly understand what is wanted of them, since they have completely lost their grasp on their business affairs.

Generally there is a besotted, reckless interest in the coarser pleasures. The patient eats carelessly and in unbelievable quantities; he drinks and smokes so long as there are any materials within his reach, heedless of all results of his intemperance. His thoughts and wishes finally all center about the matter of eating (as the following abbreviated but literally reproduced letter shows):

"In the letter which I am writing I request you to send me cigars, 100 appels, 10 Bologna sausage. You have not sent me this I have no money and so can buy nothing . . . dear, send me a liverwurst and and 1000 appels and pears, 1000 potatoes and

100 pieces of wood and logs and 100 cwt. of coal . . . send 100 apples I have appetite for such things, send me 100 cakes of chocolate I have appetite for chocolate send me a tart I have appetite for tarts send me carrot-tops I have for carrot tops send me 10 cakes, round cakes, cheese cakes, almond cakes, sweet almonds and a plum cake and a chocolate cake, send me a nut cake and an appel cake and pear cake, and a pear cake, 1 pear cake, a nut cake, 2 apple cakes, 10 apple cakes, 20 apple cakes . . . send 10 Bologna sausage, 10 liverwurst 10 blütwürst, 100 apples, 100 pears, 1000 cakes of chocolate, send me a 100 butter cakes . . . send 1000 million pan-cakes, 2000 cakes (the number is 100000000) 1000 carrot-tops. 1000 peaches send 100 nuts, 100 apples, 200 pears, 300 nuts, 400 tarts, 5000 100 cakes of chocolate, 200 apples, 200 pears, 300 cakes of chocolate, 500 apricots, 600 oranges, 70 lemons, 80 peaches," etc.

Besides the many omissions, the repetition of words and letters and the dearth of punctuation marks, one notices the great laboriousness and monotony of the content which brings in an endless repetition of the same ideas and wishes, also the increasing size in the figures which reach quickly into the millions.

Sexual excitability is often increased even with lost potency. The patient addresses obscene remarks to the maid-servant, makes coarse sexual advances to his own daughter, his neighbor's wife or to a little girl, masturbates before his children or in public. Women patients write love-letters to people in high position, are gushing in manner, lift their clothes, undress before men, prostitute themselves recklessly.

In the further course of the disease a very characteristic childish euphoria develops which is evident in the happy laughter, the friendly mien when talking, and in the hearty greeting of entire strangers. In spite of the rapid mental decline everything with him is "faultless"; he is always "pleased," everything is "beautiful," "fine," "colossal"; he is delighted at being brought

to the institution. In other cases, on the contrary, there is a kind of general idea of the severe change which is coming over the personality. The patient feels mixed up in his head, that he has some brain-sickness; he thinks "I am surprised that I know when I was born." He does not wish to remain in the world, tries to shoot himself, to spring from the window, holds his head under the water when in the bath, although his attempts at suicide usually miscarry on account of their being poorly planned and half-hearted.

The patient's ability to work is soon affected by the advancing dementia. He begins to be disorderly and careless in his ordinary occupation, neglects his working hours, important commissions, goes to the wrong place to work, wishes to go to his business at night, loses or mislays valuable objects, money, papers, either neglects his work entirely or does it at unseasonable times and gets into difficulties by the most inconceivable errors, makes mistakes in spelling, gross failures in his accounts, omits the heading to his letter without noticing it. A surgeon made out death certificates on envelopes; an instructor in a speech before the school repeated the same sentences over and over. An officer thought the orders of his superior must have become more and more obscure and illegible since he could not memorize them as formerly.

Usually the patient ceases to concern himself regarding his obligations. He evolves all kinds of impractical and preposterous schemes, which plainly reveal his loss of intelligence and judgment. He throws food into the toilet, wears his clothes hind side before, forgets to put on his shirt when dressing, cuts the buttons from his coat, paints his wagon sky-blue, changes his furniture all about, walks into strange houses and attempts to go to bed; he draws the corks from bottles, lets water run in his room to bathe, runs naked into the street with a burning lamp, gets into the tub with his shoes on, climbs trees in the park, bathes in the

river in the center of the town, opens his valise on the street. One patient gathered pieces of ice to place in his stove as coal. In travelling he gets into the wrong train, fails to buy a ticket, does not reach his destination, loses his baggage and his money, is picked up wandering about destitute or begging. One patient was found on the street where he was digging himself a bed in the snow because all the hotels were full. He buys up all kind of articles, trombones, cigars, canary-birds, applies to various lodges as a member, runs into debt, makes false change, squanders and gives away money and property.

Often he comes into conflict with the public regulations or with the law. He sets fire to his bed, fires off his revolver to show it is not rusted, runs about on a railroad embankment and is injured by a train; he digs holes in the gutter, cuts down trees in the public garden with the idea of transplanting them. A patient gathered his neighbor's crop of potatoes and planted the stalks between those of his own so that they also were ruined. Crimes against property are very common. The patients are guilty of fraud in frequenting hotels without having money; they order whatever they please for themselves and others, help themselves to anything that pleases them in the shops, stuff their pockets with whatever comes to their hands. One patient searched the till in a store and emptied it before the eyes of the owners; another tried to catch the swans in a public park. A post-man opened a package containing edibles and consumed them; a train-conductor went into a market and took a sack of apples. One man tore a pork sausage from its nail in the presence of the merchant, took it home and put it in his cellar; another loaded himself down with worthless empty bottles; others steal bicycles, crosses from graves, sacred statues. Women neglect their cooking, throw soda or sand in the soup, spoil the food, stay up at night to make coffee, cut up dress goods, bring home useless articles from the bargain counters; one patient enticed children to her in order to cut their throats.

The behavior of the patient usually very soon betrays the annihilation of the mental personality. They are quite unable to care for themselves, soon lose interest in outward appearance, do not wash themselves, do not concern themselves about meals. They undress before their children, urinate in their room, play with their genitals, grab food in their fingers, allow themselves to belch loudly, smear themselves with feces. Finally they become volitionless, good-natured, tractable, blankly indifferent to everything about them. In other cases one encounters a peculiar repellant, unapproachable attitude. To all remarks the patients give angry retorts without apparent reason, without appropriate affect and uninfluenced by friendly persuasion; they struggle against well-meant attentions, show senseless resistance, cry and grunt.

Finally, however, there are some patients who maintain good outward behavior in spite of the deepest deterioration. We see a patient who greets us politely, keeps his apparel in order, has his pillow arranged with painful exactness, but who has no idea where he is, hardly recognizes his relatives, can give no account of his past. In such cases since the patient ceases to complain, sleeps much, shows a good appetite and takes on weight, his condition is often not appreciated by others until the dementia is very far advanced. "He still knows every one," the relatives insist, if the patient recognizes his wife or happens to recollect that he has children. A patient was brought to me who was still filling the post of cashier while he was already frequently soiling himself and could not add simple sums. Another, a physician, came himself to the hospital directly from an extensive practice in order to have an operation for paronychia. When he became confused in the night and got into the woman's division by mistake it was found that he was already much demented and did not even know the dose of morphine.

The demented form comprises about 53 per cent. of my

paretics in Heidelberg, although these patients on account of their milder symptoms do not come so often to the institution as the other forms. In Munich, where committment to a hospital is made easier, the number of the demented form reaches 56 per cent. of the men and 73 per cent. of the women. Junius and Arndt found 37.25 per cent. and 40.93 per cent. respectively, in Dalldorf. Naturally the distinction is fairly arbitrary. The female sex seems to favor this form; the same is true of juvenile paresis, which is nearly always of the demented type. The earlier ages seem to me to be more frequent, since over 55 per cent. of my patients had not yet passed 40 years, against 52.2 per cent. of cases of paralysis in general. The average duration is given by Junius and Arndt as 30.05 months in men and 26.4 months in women, or somewhat longer than the average for all paretics. Of my patients 44 per cent. died within the first two years. The frequency of convulsions was greater than in any other form, while remissions were relatively rare.

In the disease picture of demented paresis the two groups of psychic disturbances which in more exaggerated form typify the other common varieties of paresis, namely the depressed mood with ideas of smallness and the expansive excitement with grandiose delusions, present themselves in clear form.

Depressed Form.—The depressive form of paresis is characterized by depression or anxiety with delusions, which may accompany the whole course to complete dementia. The depressed mood has its origin in the initial feeling of illness which is experienced, with which are associated the general, previously described indications of a gradually progressive weakness of memory and intelligence as well as an increased dullness and lack of volition. The patient's mood is expressed mostly in hypochondriacal ideas, his health has been ruined by masturbation, he is syphilitic, his insides are bad, he has had a stroke; he has the brain of an imbecile. Disease of his blood-vessels has developed

because he once sat down on a blood-sucker; the skull has become soft, is swollen in places, the brain burns, is drained off, "like a swamp," the nerves are over-stimulated by the thoughts. Usually there are numerous uncomfortable sensations in various parts of the body, twitching, burning, pulling, sticking, which vary frequently and may be influenced by suggestion. The abdomen is swollen, is like a rock, the head is half paralyzed, there is a crack in the neck; something is wrong in the brain. The patient consults a physician for all sorts of minor troubles and on account of neglect or lack of observation of the objective signs a diagnosis of neurasthenia, hysteria or hypochondria is made.

The complaints of the patient become more and more nonsensical. They have no nose, no eyes, no liver and no kidneys, they have two bodies; the stomach is bursting, the esophagus and the bowels are stopped up, sewn together, closed by a cork, so that they can neither eat nor evacuate the bowels; there is an attachment between the palate and the brain. The skull is empty, the larynx is ruptured, speech is impossible; the intestines are spoiled, blocked by germs. Everything collects in the abdomen; the stomach, the mattress is filled with urine; the food goes up into the head or passes directly through them; the lungs are gone; the legs are of ice; everything runs out of the mouth like saliva. There is music in the belly, everything is turned to chalk, filled with odors. The patient has no more blood, is composed entirely of poison, is decayed even to his bones; he has all turned to pus; he infects all who come near him, exhales a musty, rotten odor. In his belly sits a moloch who crawls about and devours him; fire burns inside him. The hands are smaller; the head is shrunken, changed about or lost, the tongue frozen, the abdomen bloated. The skin is covered with paste; there is a crust of dirt and lice all over the body; abdomen and teeth are gone; the hair has fallen out. Arms and legs have become enormously long, the ribs are as large as a giant's, the ears are of wood, the tongue of gold; 3,000 marks are

sticking in his side; a 100-pound stone is lying on his breast. His whole person has become doubled, four-cornered, changed into a horse, invisible, only "so large," weighs only twelve pounds; he has only eight breaths left. He no longer has any life, is already dead, is in the churchyard, is "a long time deceased," buried, a "living corpse," the "eminent departed," he has no name.

The delusion-formation may extend to other persons. His wife cannot appear, is dead, incurably ill, insane, must be treated; the child has no head. In compliance with these "micromanic" ideas the patient makes spasmodic attempts to force something through his closed-up esophagus, presses and squeezes his neck, keeps his hands constantly on his tongue, his anus, his genitals, sits for hours on a commode chair; he resists anxiously any change in his position because he cannot move his enormous hands, or because his tiny legs must crumple beneath the weight of his great "chanticler-head."

Associated with the hypochondriacal ideas there are often delusions of sinfulness which sometimes dominate the picture. The patient has not done the right thing, has lied, has behaved improperly; he blames himself for the death of his wife, accuses himself of making false entries in his books, of commiting perjury; he deserves to be thrown on the dung-heap, to be tortured; one of my patients informed the prosecuting attorney that he had stolen. The pastor in his sermon makes reference to the disreputable life he has led; the police will come, he will be arrested, locked up, hanged, burned, put in a sack, shot to death; his limbs will be cut off, he will be beheaded; the scaffold is erected. Everyone despises him; so he only gets salad when others eat cutlets. He is the most unlucky person, is ruined, brought to beggary; he has made a mess of his life; nothing further offers, everyone must go hungry, must be slaughtered.

Persecutory ideas may comprise the whole content of the

depressive delusions. They are often accompanied by hallucinations of hearing. People stand outside and make note of everything; they call him wretch and scoundrel; "he is not here yet, the old blackguard!" his children clamor; the magistrate has ordered that he be shot; "he has syphilis." Some one plays tricks on him and gives him electric shocks, ruins him, throws poisonous vapors into his room, breaks bones in his abdomen, puts poisonous crumbs in his food; he feels them rush up to his head. The devil speaks to him, threatens him, he is himself in hell, the prince of hell; the end of the world will be to-morrow. Soldiers come in the night, black devils, ghosts, women in white, policemen, men who wish to murder him. Giants dance about; bears bite at him, lions come at him; a crocodile glides between his legs. These latter hallucinations are suggestive of those of the alcoholic and are perhaps colored by the influence of that agent. The same is true of the ideas of jealousy which sometimes arise.

The mood of the patients is often one of striking indifference, in spite of the disturbing nature of the ideas. They relate their delusions in a monotonous, dull manner without seeming to be affected by them. Now and then there is an abrupt changing about of the mood, sometimes in contradistinction to the expressed ideas, sometimes upon the appearance of grandiose delusions; the patient thinks he has lived 4,000 years, an eternity, that he is the first prince of Clovis, is about to grow a foot taller, has broken the bank at Monte Carlo, wishes champagne to drink. On the other hand, very severe states of anxiety often develop. The patients are much disturbed and confused, mistake their location and surroundings, fall into a state of utmost desperation. They glance about in terror at every noise in their neighborhood, tear the clothes from their bodies, struggle blindly with all their strength, do not remain in bed, slip under the bed-stead, crawl about, kneel, pray, whine, beg for mercy, jabber, gesticulate, mumble unintelligibly. Others wander aimlessly about half-clad

or naked; or they lie in bed trembling and sweating with the covers drawn over their heads offering a blind resistance to every approach from without. They cannot be brought by any means to the observance of the simplest regulations, so that going to bed and arising, dressing and undressing are accomplished only after desperate struggles. Many patients hold themselves absolutely rigid, are mute and non-coöperative; others utter inarticulate cries for hours and days, or they shout monotonously the same isolated words: "Ach, ach, oh, oh, oh," "Ach Gott," "poison—poison," "our death."

Sometimes in these states the patients are very excited and violent. They break out in confused abuse, are ugly, make sudden, reckless assaults, try to drag other patients from bed, to strangle them. The relatives may be especially fearful that the patient may obtain a revolver or a knife. One of my patients in his anxiety wrecked his whole room and would have killed his wife, whom he took for a burglar, had someone not rescued her from him. Often enough there are actual, but usually weak or senseless trials at suicide or self-mutilation. I have several times seen attempts to cut off the scrotum or penis. One patient shot himself in the hand; another struck himself on the head with a hammer, others run against the wall, jump from the window, or swallow large objects in suicidal endeavor; in the intestine of one patient I found a thick wooden cigar holder and two screws several inches long. One patient went to the cemetery to buy himself a grave. The duration of the severe anxiety states varies between hours and weeks. Not infrequently the condition ceases suddenly only to begin again as abruptly or to be lost in a change of mood.

Often in the course of paresis we observe long-continuing stuporous states, which perhaps may be mentioned at this point. The patients speak neither voluntarily nor in reply to questions, lie motionless, taking no part in their environment, eat no food,

wet and soil their beds. Urgent commands are either not obeyed or complied with slowly and hesitatingly. The mood is usually one of indifference, but often tinged with anxiety or depression. The grasp and orientation as a rule are disturbed, but generally correct themselves, although this is hard to determine, as the patient does not speak at all or only in a whisper. Delusions and hallucinations may or may not be present. Many patients show prolonged or transitory catatonic symptoms. They are cataleptic, with echolalia and echopraxia, make monotonous movements, waving the arms, snapping the fingers, singing or repeating the same word incessantly. They drool saliva, retain urine and feces, assume uncomfortable attitudes, keep the eyes tightly closed, do not resist when pricked, press the lips together when a spoon is held to the mouth, do not reply to questions and resist every attempt at movement, without, however, any indication of anxiety.

In a small group of cases we meet a complex of long-retained persecutory ideas, especially the delusion of bodily influence by electricity, mirrors or poison. Besides very marked pains and unpleasant sensations in various parts of the body, there often occur hallucinations of hearing. Where we are dealing with true paresis and not with a syphilitic psychosis, these symptoms are usually referable to a previously existing tabes.

After careful classification of my material in Heidelberg the depressive form constitutes 12 per cent. of the cases of paresis; in Munich the percentage seems to be slightly lower. Junius and Arndt give 16.8 per cent. of men and 19.47 per cent. of women to the depressed form. This variety seems to have a slight preference for the later years of life than the average of all cases. Convulsions are somewhat less frequent. The duration is held by Junius and Arndt to be somewhat shorter than that of the demented form; it is given as 23.7 months in men and 20.4 months in women. According to my figures, 58.6 per cent. died within the first two years; this form is therefore to be regarded as a severe one.

Expansive Form.—Expansive paresis either shows expansive excitement from the first or commences with a more or less definite depression, which is sometimes simply a slight reduction in mood with anxiety, sometimes reveals the features described as depressive paresis. The further development consists in a gradual, although sometimes rapid disappearance of the depression and feeling of illness. The patient becomes tractable, happy and voluble, but betrays by his poor realization of his condition and situation, as well as by his loss of judgment and discretion, that there has been a change but not an improvement in his condition.

Very soon now there develop the characteristic ideas of greatness, the "megalomania" which is the best known clinical picture of dementia paralytica ("classical paresis") and constitutes the popular conception of the whole disease. Its content takes in all the patient's associations, his mental and physical capacity, his knowledge, his external relations, his property, his future. At first perhaps the ideas of greatness remain within the bounds of the conceivable and possible and have the appearance of simple childish prevarications. The patient feels stronger than he ever has, is extraordinarily well preserved, a brave fellow, superhuman, very well-educated, was smarter at the age of 16 than a professor, writes a wonderful style, understands many languages, although he cannot speak them at the moment on account of having lost his teeth; he has a marvelously beautiful daughter. He composes excellent poetry, has an incomparable voice, an iron will, a moustache like the kaiser's, great sexual power, high connections, great prospects, associates only with fine people, drinks champagne, is highly esteemed, can make a good match any day, enjoys the highest confidence of everyone. His business goes splendidly, yields a large profit because he sells goods of little value at high prices. His employees have all founded enterprises of their own. He will start coöperative concerns which will win

first prize, will build cheap houses for laborers and policemen, will make important discoveries, will hold public lectures, give concerts, educate the people, write a book which will create a great sensation and bring him large sums; he will break the bank, build himself a castle, undertake wide travels, give great feasts, will allow himself to be elected to the reichstag and without doubt will be soon chosen for a ministry; he has an enormous inheritance in prospect. At school and at various universities he has astonished his teachers by his talents, has passed his examinations "summa cum laudissima," won a large number of prizes, is the holder of numerous patents, master of all the arts of chivalry, the pet of all the women, saw service in the wars from 1848–70, captured Napoleon, has been in Morocco, has travelled all over the world.

In all this the marked mental weakness of the patient is all too apparent in the inconsistency of his delusions, in the dreamy artlessness with which he builds his air-castles and in the lack of judgment in the face of obvious objections. A poor parish clerk told me triumphantly that he would demand 1,000 rubles for every day of his incarceration in the institution and then with this money would live magnificently and in freedom. Others delude themselves with plans to sell from now on all goods at 50 per cent. profit, to make millions from post-cards with poetry on them, or to buy up all the lottery tickets so as to be sure to get the grand prize. A good glimpse into the stream of thought of such a patient is given in the following letter:

"O God, O God, I have so many ideas, an idea every second; I am indeed insane—my poor head! I am the greatest genius that has ever existed and am sitting here in a mad-house; I, poor wretch, I am capable of anything; let me go to my poor wife; I am an officer; they do not dare detain me; I have fought in the war; I have to keep doing general utility work although I do not desire it. I give away my best ideas; literature and philosophy

are dear to me; I cannot estimate the value of all my patents; I think out a new one every quarter-hour. Do you wish to procure a horse and carriage, doctor? I am the best judge of horses; I will build you the most beautiful bicycle in Europe; I am eternally obliged to you; you are my deliverer, my savior; in me you have rescued for the world a genius; make me well; I kiss your feet in thankfulness; God stand by me, deliver me from this mad-house; crush these people who are so persecuting me. What a fearful place this is; the architect knew nothing! Look you, doctor, I will show you how to alter it. The building is much too acoustic; they must have felt carpets. We will change the insane hospital into a castle. I will re-construct it; I am acquainted with historical matters. We make the excavation like that of Schliemann—ach Gott, is his name Schliemann? I lose my memory; I am insane, I am delusional; give me prussic acid so I may die; I will gladly die. Let me free. I cannot spend my life in a mad-house; what will become of German science, of German universities! I am a genius, as you must realize; I speak French—am I then insane? But it is a blessing that I came to an insane asylum; shall I recite Faust to you?" etc.

As a rule the senselessness and fantastic nature of the grandiose delusions increase quickly and without remissions. The patient thinks he possesses extraordinary physical strength, can lift ten elephants, is 800 years old, 9 feet tall, the most beautiful Adonis in the world, weighs 400 pounds, increases 25 pounds every week, has an iron chest, sinews like a man-eater, an arm of silver, a head of pure gold, travels a thousand miles a minute, can fly. He is infinite, has died and again come to life, can have intercourse with 100 women, has 1,000 million boys and girls, a compressed brain, has run a race with the grand duke. His urine is Rhine wine; his evacuations are gold. Ten years ago he had an enormous chancre, his sexual organs and fingers are constantly getting larger; his brain is still growing; he has an immense

movement of the bowels. He has studied all sciences, speaks all the languages in the world, plays Wagner at sight, impersonates Don Carlos like a God. He is the greatest physician and pharmacist, the most famous opera singer, can cure all diseases, awaken the dead, has learned the speech of the Brahmins and Buddhists; he makes wonderful monograms, can drink a hundred measures of beer a day, sing 1,000 solos, can make the weather, wreck the whole universe; sun and moon listen to him. In his walks he repeats the Lord's prayer 500 times, confesses every two months, is very pure and therefore sure of reaching heaven.

Furthermore he is "his excellency," count, prince, commissioner of forests and officer, president of the reichstag, adjutant general, general of the Ulan guards, admiral, commandant of militia with rank of general, unanimously chosen the white emperor, kaiser and sultan, Bismarck, emperor of Japan, China or Russia, shah of Persia, Napoleon, the greatest man in Germany, pope, ruler of Europe, Kaiser Wilhelm XVII, Messiah, field marshal, owner of the world. God Almighty, Super-God. The pope and the archbishop of Cologne are his cousins; his wife has married the king; God is his uncle; his children married counts and princes; his son is king. His mother is 101 years old, fresh as a rose. He keeps a mistress, six girls, will have more later, has a countess for a wife, a golden wife, 30,000 wives, 70 children, who are all in Africa, millions of children, beautiful as angels, he has married a queen with 50 million dollars; all women are attracted to him. His wife earns great sums as a prostitute; his fortune runs up to 40 millions bringing 20 per cent., even to 10,000 billions and trillions. He is richer than six kaisers, receives a daily increase of two millions, interest of nine millions, in 80 years 10,000 millions. He owns all banks, ships with tons of gold, a great block of gold in South America, gold-mines, a house of 100 rooms, a million horses, 1,000 oxen, 1,000 automobiles, 120 elephants, countless canary-birds, nose-glasses made

of rubies, 1,000 gold-spectacles, 1,200 large books, a gold carriage. Enormous hunting-preserves belong to him, 600 trotting horses, herds of cattle in marble stalls, 100,000 ships, each 100 feet long and 100 feet wide, with 10,000 electric propellers. He has a property in Heaven, a place of honor at God's side; God talks with him, electrifies him. Billions of laborers work for him; his land bears twelve times a year; he receives 100 billion of telegrams every day.

A statue has been erected to him, the Kaiser has ordered him to Berlin as major-general, the grand duke presented him with nickel-plated horse-shoes, the pope sent him 500 workers. He was general for 6 years, commanded a million soldiers in the war, blew up 70,000 men with bombs, held parade before the Kaiser; in company with a baron he shot 200 animals at one hunt. A hundred years previously his head was cut off; he has shot himself; has taken 20 grains of arsenic, has made a mountain larger, was in America, Jerusalem, went all over in his own warship, sang the Lohengrin, built a great city with famous picture galleries, secured Siberia from the emperor of Russia, married all the Bourbon princesses. The Savior passed the cup to him, God favored him with revelations. The evening star tells him everything.

The patient is now Chancellor of the Empire, lieutenant with a salary of 10,000 marks, he will enter the cuirrassier guards, lends gold by the billion, opens 1,800 marks worth of champagne; an English lord brings him millions; to-morrow he will receive 70,000 marks. He will build a villa and cities, a church, a menagerie and a factory, will establish a wine industry, will erect a hotel worth 10 to 12 millions, buy a steamship, steal an automobile, order a special train. He will take a drive with outriders, will arrange a parade of the most distinguished people, will travel to Rome to the pope, to Australia, will shoot deer, pheasants and lions; he goes on a whale hunt, captures animals by glue on the

trees, will fly to India, to the moon and stars in a machine, will hunt diamonds, make a study of sun, moon and polar-bears, will preserve the North pole with warm water and the South sea with ice, with a steam plough turn the whole of Bavaria into a vegetable garden for everyone's use, pay all the taxes of Munich, wash all negroes white with benzine. He will build a bridge over the ocean to India, erect a tower in a garden which is a thousand miles long with a golden roof and its own theatre and circus; he will invent a flying machine and fly around the universe, will dig a tunnel through the earth to California. He will marry a wealthy woman, the empress, a very beautiful woman with large breasts, will have a celestial wedding and travel alive to heaven with music. He slew all the English with gold balls, burned down 14 houses; in the future all people will be named Müller and numbered 1–100,000.

Women have jewels worth 60,000 marks, rings, a handkerchief studded with diamonds, silk waists and wraps, millions of necklaces, a glass-enclosed theatre, a castle in Corfu, arrive "like a fairy," eat from golden vessels, weigh 800 pounds, spread a piece of bread with five pounds of butter and 40 pounds of caviar, kill every day two calves, two cows, two sheep, two geese, two chickens; one patient told of a gold douche-syringe studded with jewels. The patient has inherited 100,000 marks from her stepmother, has won billions at cards, gives brilliants and jewels to the needy, comes from the deity, is a sister of the kaiser, empress of Germany, first woman doctor of the world, mother of heaven, last daughter of Archangel Gabriel. She has led a loose life, has had intercourse with counts and barons, king and kaiser were there; the pope gave a benediction. Her husband is a general; she will marry Jesus, King Ludwig, she has 600 children by him; her betrothed is a millionaire, "so insane that he is senseless." Her sister is wedded to a rich French nobleman. She will marry a young man, will have a civil marriage this afternoon, is preg-

nant, will give birth to three kaisers, twins and triplets; she has 600 virgins for daughters, must still make many children with which to fill heaven; the Kaiser will be godfather; at Christmas she will again be a virgin. God speaks to her in the storm, travels with her to Berlin; every night she travels eight goose's flights in the sky. She was in Venice and Jerusalem, gave a window to the church, has appeared as Clara Ziegler; she will open a salon, heal cancer in three days, make wine from stone, make all men happy, erect a sanitorium, a home for friendless, found fraternal orders for everyone's good.

Although the fertility of these delusions varies greatly in different patients, some may be unable to formulate new ideas; they endeavor to invent new expressions to convey the one idea of the greatness of their possessions and happiness; money plays no rôle; money flies from the pocket; the patient "owns practically everything"; "100,000 trillion billion, onen, oten, tatten, satten, trillion, tater, tatter, taller" said a patient who could find no words to give a conception of the immeasurableness of his riches. "Words cannot express it" thought another. Usually current events as well as personal relations and interests are reflected in these ideas but always in a distorted manner. It is noteworthy that, as a rule, the grandiose ideas of the women keep within moderate limits and do not transcend the possible to such a fantastic degree as those of the men.

The consciousness of the patient is usually slightly clouded during the development of the delusions of greatness. The environment is grasped only imperfectly and fragmentarily. They may have no clear conception of time, place or circumstances as may be shown by testing. They are little concerned by practical matters but are much more engrossed in their visionary enjoyments and plans. The train of thought is usually lacking in cohesion and is easily influenced by outward occurrences. The moment anything is suggested to them, the greatest diversity of

ideas follow each other in profuse variety, poorly worked out and full of most obvious discrepancies. Occasionally some details of the delusions are long held, but usually all are quickly forgotten or replaced by others. In most cases it is possible by suggestion to induce the patient to enlarge and embellish his ideas of greatness, to relate the most fabulous adventures and hazardous projects. Often, as in the example given, there is distinct flight of ideas. The increased distractibility is clearly noticeable in the writings, the relating of their desires, commands and plans.

In some cases there are transitory hallucinations of sight and hearing, but these usually play but a slight rôle in the symptom picture. They consist usually in disordered imagination. One of my patients, an artist, in the beginning of his disease heard all the details of a sexual embrace of one of his girl students in an adjoining room; others hear commands from God, voices which "speak good of him."

The mood of the patient is a happy one in accordance with the content of his delusions, he is self-conscious and full of confidence. Often he reaches a state of most extravagant felicity. The patient with tears of joy gives thanks to Heaven that such happiness has come to his lot. He would embrace the whole world and bless it as he himself has been blessed, now that his fate has been so magnificently ordained. Everything about him is unsurpassed and costly; his meals, his house, his clothes are fit for a king, the waiter is a genius, his friends and acquaintances are excellent, honorable, distinguished men, his children, finished products of good breeding and intelligence. Now and then through the elevated mood, there glimmer faint indications of an understanding of their condition, an admission that they are somewhat nervous and in need of rest. "My brain is enormous but it has its limits; I crave a day's pause," said one patient.

At times there are marked hypochondriacal ideas mixed with grandiose delusions. The patient is insane, has no understanding,

has worms in his head, no brain, must get a new one. The blood is dried up, the larynx gone; he cannot swallow; the head has become too small. The neck of the bladder is severed; he has no abdomen; his posture is altered, the body corroded, the spinal cord removed, the sexual organs are changed; he has an iron rod in his body, a human head in his belly, two children inside of him. Someone has poisoned him with prussic acid, has cut off his penis, torn his arm off, will stab him in the eyes; he will die to-morrow at 10 minutes past 11. Depressive ideas of other sorts are sometimes seen; the patient has committed many sins; he will be injured; a catastrophe is coming; the kaiser is dead; he may be shot or stabbed or have his eyelids cut away. The mood may thus be suddenly changed to one of deepest depression or great anxiety with whining and crying. This rapid change of disposition is plainly shown in the letter given. As a rule the reversals of mood do not last more than a few hours or days, less often they occupy a longer portion of the disease-course.

On the other hand there is frequently a marked excitability. An expression of doubt or an argument against his expansive notions easily produce violent but quickly fading anger, especially if he can think of no reply. He threatens wife and children with knife or revolver, attempts to strangle them, breaks the windows, pounds the doors, overturns the furniture or throws it from the window. He gives orders to those in charge, demands stormily of the physician that he be released, spits in his face, accuses him of robbing him of his freedom, after having perhaps a short time previous offered him a professorship or a woman worth 1,000 millions. He also sometimes shows reckless violence toward other patients, since he has not the slightest understanding of their condition, but thinks they are perfectly well and are merely impudent swindlers. He writes the state's attorney complaining of them, invokes upon them the punishment of hell, threatens to annihilate them with his artillery, to throw them all

in chains, to have them "beaten by 100 black savages with iron whips."

In the psycho-motor field the patient almost always shows a certain restlessness, which under some circumstances may increase to a very severe grade. The patient is unstable, busy, full of undertakings, neglects his work, wanders about, drives all day in a cab, travels in luxury, makes acquaintances everywhere, visits strange people, invites all sorts of persons to his house, takes all the neighbors' children to the theater, behaves peculiarly, is noisy, talks much and loudly, writes numerous letters, dispatches, poems, falls easily into a dispute, begins to drink, to chew, or to take snuff excessively, commits sexual irregularities. At the same time he begins to attempt the realization of the great plans which originate in his feeling of unlimited capability and excessive desire to do something. Without any consideration he embarks on the most various undertakings, which not only exceed his knowledge and means but even surpass the bounds of possibility.

Usually the schemes are dropped after a few foolish steps have been taken, because the idea has been replaced by a new one. He suddenly enlarges his business, gives his employees absurdly high wages, goes to America to enjoy himself, begins to build, makes a very unsuitable marriage, asks for the hand of his house-servant, sues for divorce in order to make a distinguished match, shows in his conversation his infatuation for some rich heiress, telegraphs to the crowned heads requesting high orders or titles, appears at the royal castle for an audience, makes large gifts, fills the papers with senseless advertisements. In the same way he replies to everything offered in the periodicals, parrots and cooks, buggies, bicycles, rings, country-houses, and partners in marriage; he purchases anything he happens to see. He orders absurd quantities of the most varied articles, which he expects to use in carrying out his plans, canary-birds, watch-chains, fur-

niture worth 1,000 marks, automobiles, cloth to the extent of 50,000 marks; he writes checks for millions. One of my patients who was rich and an enthusiastic photographer, sent a telegram ordering 200,000 marks worth of pyrogallic acid for his use. It can thus be understood how a patient can so very quickly squander large sums of money, get into serious difficulties and cause serious consequences both to himself and to his relatives.

In all the excited and senseless actions there is plainly to be noted that blunting of the moral sense which is the usual accompaniment of the disease. The patients are not only neglectful of appearance, unclean and disorderly about their clothes but they lose all understanding of the simplest demands of propriety, tell loose stories, relieve their necessities without heed to surroundings, urinate in the drinking cup, break wind, boast in shameless manner of the sexual accomplishments of wife or daughter, accost women on the street, show themselves publicly with loose women or try to make their acquaintance. We often see patients commit dangerous and criminal acts, careless setting of fires, small thievery, bold cheating, impostures, assaults. These things are usually done so openly that they are immediately detected. One of my patients, when on a train, seized the valise of a fellow-passenger and would have made off with it. Another on the street attempted to lead away four horses. Since he afterwards perhaps denies his deed in spite of the clearest evidence he is often held to be a hardened and clever swindler. A locomotive engineer ran through a large station at an 80 mile speed and afterward laughingly inquired what would it matter if a train ran off the track or a few men were run over; that might happen to anyone. Often the individual only reaches an institution as a patient after he has committed numerous offenses against public order or decency, resisted the police, brought his family in a short space of time to poverty, been mistreated, punished and become exhausted by dissipation.

Often there supervenes a certain quieting down, in which the patient denies some of his large ideas and plans while others seem more or less firmly fixed. Under observation in the hospital, he may, aside from a certain mental weakness, seem soon to become almost normal, but when he is given a trial at home his actions usually very quickly betray the profound disturbance of his whole personality. On the other hand the expansive delirium may continue a long time, even months or years, the ideas becoming constantly more impossible and absurd. One notices that the quickness and richness of the thought life are steadily failing. The delusional ideas become more limited, inconsistent and contradictory. The patient is "Hercules, a millionaire and a diver," "chemist, philosopher, horse-doctor and blacksmith, diplomat and district leader," "life-saver, graf Waldersee and manager of a slaughter-house," he can "pray and recite," has a beautiful blonde wife, blue silk underclothes, eats five pounds of beef, champagne and lettuce, gives each of the patients a million dollars; he pays the doctor a salary of a million dollars and expenses.

The mood is dull and disinterested and the activity is finally reduced to the chanting of senseless syllables, the scribbling of unintelligible letters and dispatches, the drawing of childish, incomplete pictures and plans, the stuffing of his pockets with a collection of all sorts of rubbish, the writing of endless rows of figures by which he expresses his immeasurable riches or earnings which he hopes to realize through his enterprise. The patient grows constantly more demented and stupid, although a dull reflection of the grandiose trend may for a long time illumine the mood background. With friendly, happy face he sits and murmurs perhaps in hardly distinguishable speech, some words taken from his exalted fancies: "good to eat," "millions," "fine horses," "golden Empress," until finally even the last recollections are lost in the complete blotting out of the psyche.

During this development, usually following attacks, there may

occur more or less acute excitements in which the patient is confused, disturbed, abusive, shouts, makes reckless assaults, undresses, runs about naked. In this condition the patients are usually practically without understanding and unmanageable. Often there is associated a monotonous, senseless restlessness, rubbing the fingers, chewing movements, kicking, rolling, rhythmical muttering or crying, senseless rhyming.

I have found the expansive form to comprise 30 per cent. of my Heidelberg cases, in Munich 21 per cent. men and 22 per cent. women, since here, for reasons already given, the demented type forms a larger number. Junius and Arndt found in Dalldorf, 26.73 per cent. among the men and 25.88 per cent. among the women were of the expansive form. In Heidelberg the women's division showed a lesser number of these cases. The earlier years seem to be slightly less affected since but 45 per cent. of my cases were under 40 years at the beginning of the disease. Convulsions are noticeably less frequent, remissions more common than in demented paresis. The duration is calculated by Junius and Arndt to be 28.2 months for men, 27.7 months for women, or longer than for the depressive and, in women, longer than for the demented types. According to my figures 40 per cent. die during the first two years, or less than in other forms. Expansive paresis is then to be reckoned as a relatively late occurring form with rather a preference for the male sex, a fairly mild and protracted course. It furnishes also a considerable number of those cases with prolonged duration and lengthy remissions which we are wont to designate as stationary paresis. One of my Heidelberg cases lasted 7, another 4 years; two others were still alive after 8 and 14 years respectively, while among twice the number of demented paretics only three cases of seven and one of nine years' duration were found.

An admixture of the depressive complex in the course of expansive paresis may assume the appearance of an independent

stage of the disease. Rather seldom there are seen frequent, irregular alterations of the two pictures or a mixed condition with each in equal prominence; oftener they follow each other as separate attacks sometimes merging immediately one in the other, sometimes with a remission between; several repetitions of these changes are often observed. This is spoken of as a circular course of paresis. The expansive picture seems, from my experience, to form, unlike circular insanity, the introduction of the disease, if one excludes very indefinite prodromal symptoms. The depressive picture as already described shows a preponderance of hypochondriacal ideas. The patient has lost his memory, has no more intelligence, his thoughts are gone; the mouth is fastened shut, the bowels cut out, abdomen full of pus; arms and legs are fallen off, the sexual organs are cut away; he has no stomach, cannot eat, carries a machine in his head; the hands are small and black; he is dead, the life has gone out of him. He has stolen, ruined the whole world, assaulted his own daughter, is the devil, is in hell, is 100 million years old. Everything is annihilated; anarchists are threatening the Kaiser; spirits are trying to kill the patient; he receives electric shocks, will be burnt, torn to pieces by dogs, murdered; there is urine in the coffee. One of my patients gave himself up to the police, many attempt suicide, one employed an attendant to look after him.

Agitated Form.—Agitated paresis is that type of course of the expansive form in which states of marked excitement dominate the picture. In this form the initial symptoms are often very slight, so that the disease seems to break out quite suddenly. One of my patients abruptly began to sing and to undress while in church. Usually grandiose ideas develop, which are even more florid and absurd than those we have learned occur in the expansive form. Within a few days the patient is glorified and healed of all his suffering and sins; everything is young; he has the Savior's crown, 124 orders, introduces a new system of reckoning

time and goes to visit the almighty God who created and eternally loves the whole world. He can make men and horses artificially, can awaken the dead, is a man of nature, king of Spain, the greatest cannonier, the world's deliverer, can transport himself with the quickness of thought to any part of the heavens. The mother of God is coming; the Kaiser has telegraphed a hundred times. He has conducted all wars, won all battles, made the greatest inventions and discoveries, has personal acquaintance with all the great men of all ages, has died many times and come to life. He has immeasurable riches, whose greatness cannot be expressed in numbers, more than decillions, beautiful horses from the sky, 27,000 carloads of gold; he builds in a moment the most magnificent castles and domes of violet moonstones, diamonds and jewels, appoints the physician emperor of the Germans, impregnates thousands of the most beautiful women with noble, divine sons.

Sometimes ideas of greatness and smallness are interwoven inextricably. The patient is desperate to think that he has allowed himself to be confined in an asylum instead of employing his millions and allowing himself to be crowned emperor. At the same time he may think his neck is stopped up and that he has countless injuries. But he will distribute among the people so many millions that no one will again speak of his insanity. Someone has designedly made him ill; his head is full of beetles but he will be born again, will receive a new brain, strong muscles, other eyes. In sudden paroxysms of desperation there may be blind attempts at suicide.

At times a confusion with flight of ideas develops, of which the following is an example:

"Das war eine Qual, in diesem Saal, nur das Knicken und das Knocken; sie haben's getan, sie haben's getun, sie haben nichts verschuldet. Nicht sie, nicht ich, nicht sie, nicht ich, nur die eine vereinte menschliche Natur, nein, nein, nein, nur die Spur, zu dem Hang, der natur . . . "

Finally the monotonous, measured utterances may resolve themselves into unrecognizable distortions of words and syllables with senseless rhyming. The patient is anxious, excited or euphoric, restless day and night, busy without interruption with his endless plans, telephoning orders to all points of the compass, writing undecipherable telegrams on every scrap of paper; he laughs, prattles, howls and sings, holds conversations with God, masturbates, is unclean and smears food and excrement about. A case of this sort is shown in Fig. 17, which also gives an idea of the activities of such a patient if left to himself in an isolated room. This man, who quickly succumbed to very aggravated symptoms, is shown in an extremely heightened mood, having torn the sheets and spread to pieces and wound the strips about head, arms and legs.

Stuporous states may occur in the course of the excitement; the patient lies stiff and mute with closed eyelids, shows catalepsy, echolalia and echopraxia or rigid resistiveness. He sleeps almost none, takes nourishment very irregularly, since he has no time or is occupied with something vastly more important; his weight sinks very rapidly. The temperature is not infrequently subnormal; I have often seen the symptoms of diabetes insipidus.

The severest cases of agitated paresis are sometimes given the term "galloping" paresis. In these we have an extremely rapid, fatal course with symptoms of excessive psychic and nervous excitement with sudden termination. As a rule these stormy clinical pictures form the conclusion of an agitated, less often of a depressed paresis, but there are cases which run this way from the first. With the rapidly increasing excitement the patient becomes completely confused and practically devoid of his faculties; he produces only a few inarticulate sounds or stereotypies, meaningless syllables, mostly in a rhythmical manner. He dances about the floor, drums and stamps with arms and legs, twists and bends himself, claps, plucks, scratches, cuts his face, blows and

grunts, will not keep his clothing on, tears, rubs, smears and spits, eats his excreta, howls, cries, strikes blindly about him. He sleeps little, takes no nourishment, but spits everything out, soils and

Fig. 17. Excited Paretic.

wets himself. The weight sinks with great rapidity, as shown in Fig. 18; the pulse is small and rapid, temperature elevated (100°–102° F.) probably mostly on account of bruises and abrasions which the patient sustains in his senseless excitement.

After a few days or weeks, sometimes as a result of an

apoplectiform or epileptiform attack, the movements of the temporarily stuporous patient become uncertain and tremulous; the mouth is dry; lips and tongue are covered with thick dark crusts; there are profuse diarrhea, cold sweat, tendon clonus, marked tendency to bed-sores and with increasing heart-weakness, per-

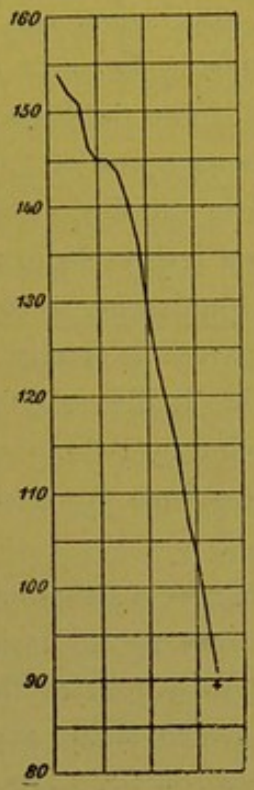


FIG. 18. Body-weight in galloping paresis.

haps after a period of unconsciousness, death occurs. It is such cases as these which were classed with certain infectious deliria of unknown origin under the term "delirium acutum." This applies chiefly to those cases in which the initial symptoms are little or not at all evident. In such, one finds widely distributed, very severe disease of the nerve-cells and infiltration of vessel-sheaths with plasma cells and lymphocytes. Sometimes one has occasion to observe the patient again and now sees undoubted indications of paresis.

The agitated form is the least common symptomatic group of paresis. It comprises about 6.3 per cent. of my Heidelberg observations; in Munich where the scope is somewhat broader we found 14 per cent. among the men and 5 per cent. among the women. Junius and Arndt give the proportions as 7.91 per cent. and 4.42 per cent. respectively. So far as a small number of observations permit of a conclusion, the earlier ages appear to furnish the greater number. Seizures are, on account of the quick course, infrequent. The duration is given by Junius and Arndt as 16.4 months for men and 10.5 months for women; two thirds of my cases died in the first two years, a large number, especially of men, within a few weeks or months of the first indication of the disease. Remissions seem to be frequent. As a matter of fact, agitated paresis is merely a subdivision of the expansive form, from which it differs only in its stormy course. Galloping paresis was found by Behr in 9.2 per cent. of the men and in 2 per cent. of the women, by Buchholz four times in 335 cases, or 1.2 per cent.

In conclusion, we have here a symptom-picture which does not occur independently but may appear transiently in all forms of paresis, especially in the early part of the course. I have seen certain delirious states which show a great similarity to the delirium tremens of the alcoholic. The patient early loses his orientation, falls into a peculiar restlessness with occupation delirium, acute hallucinations, especially of vision, sleeplessness and marked tremors, with a half anxious, half euphoric mood, but without the characteristic humor of the alcoholic. After a few days or weeks there is a quieting down and the patient becomes clear. One as a rule regards these states as alcoholic and attributes them to previous excessive drinking. For a number of cases this conception is the correct one. Nevertheless, I have, to my surprise, encountered this symptom-complex in some cases in which the patients were very temperate livers. It appears there-

fore as though there is a delirium in paresis which is very similar to that of alcoholism but still is not identical with it.

Course

As seen from the previous detailed descriptions, the course of paresis is, in general, made up of a sometimes unnoticed preliminary stage and a period of marked disease symptoms, to which the end stage of deep dementia, to be described later, is added. It is often very difficult to establish the actual beginning of the disease with certainty. A year, even many years before the first indication of the beginning of the disease one not infrequently observes suspicious changes, whose significance is only later made plain. Such are transitory double vision, fainting attacks, loss of speech, giddiness. In two of my cases there were six years between such paroxysmal attacks and the outbreak of paresis. Thomsen[1] has seen cases in which pupillary rigidity, oculo-motor paralyses, aphasic or epileptiform attacks or disorders of speech were observed 5, 7, even 10 and 11 years before the beginning of the disease itself. Junius and Arndt also record a number of observations with fainting, spasms, apoplectic strokes, a correspondingly long time before the onset of paresis. Since the disease is probably a long time in preparing itself, the possibility is by no means excluded that these are really its first manifestations; obviously they may also be a light form of syphilitic brain disease.

Still more difficult is it to determine the real beginning of the disease when years of all sorts of psychic disorders, excitability, inability to work, mental weakness, disturbance of sleep and anxious states have preceded, since these are often found in psychopaths but also may belong to the early stages of paresis. If one refuses to accept a "syphilitic neurasthenia" it will often be impossible to set the bounds for the beginning of the disease.

[1] Thomsen, Allgem. Zeitschr. f. Psychiatrie, LII, 889.

Great difficulty attaches to the judging of the relationship of earlier psychic disturbances to later appearing paresis. Neisser as well as Junius and Arndt have recorded such observations. In many cases a close relationship may be summarily denied, as in attacks of manic-depressive insanity; at most, a manic excitement may favor the acquirement of syphilis. However, it must always be remembered that manic or depressive states often usher in paresis and a remission may follow the first outbreak, which in some cases lasts years. Although, when there is an absolutely normal interval of eight, ten or more years we may exclude with tolerable certainty a connection between the paresis and the earlier disorder, still with shortening of the interval the answering of this question becomes always more difficult. It is necessary, in the first place, to ascertain whether the earlier affection presented any physical or mental symptoms which could be associated with paresis, and whether, in the interval, there were continuous or transitory disturbances which might speak for the continuous existence of this disease process. In general, the occurrence of other mental diseases with paresis is so rare that it must be regarded as purely incidental and by no means indicates a tendency to paresis through the predisposition to other forms of insanity.

The actual beginning of paresis consists, as a rule, of rather indefinite symptoms, which are usually interpreted as nervousness. They are apt to be excitability, a hasty, excitable, irritable or whining manner, anxiety states, absentmindedness, fatiguability, mental dulness, forgetfulness, a tendency to sleep or persistent sleeplessness. Accompanying these are headaches, migraine with scotoma and vomiting, giddiness, cardiac palpitation, excessive sweating, rheumatoid pains, numb feelings, muscle-twitching, writer's cramp, brief weaknesses, abdominal cramps, intestinal obstruction. In the further course the mental and physical symptoms may progress collectively or individually with very irregular rapidity. Thus there are cases in which even severe

disturbance of speech and writing with tabetic signs and lost reflexes may exist for a long time before any marked involvement of memory or intellect can be discovered. On the other hand the mental symptoms may be fairly prominent, while physical examination reveals, at first, only indefinite and non-characteristic changes. This fact furnished a wide opportunity for errors in either direction, before the cytological and especially the serological tests afforded us a means of verifying our opinions even in the earliest stages of the disease.

The sequence in which the physical and mental symptoms appear also shows great variations. That some patients retain ability to calculate extraordinarily long is well known; others preserve time orientation in an unusual manner. Often the speech and writing disorders remain very slight even into the late stages, or the pupils continue to react well; in one case, which is to be mentioned later, I saw pupillary rigidity appear only after the paresis had existed seven years, a circumstance which had led to a wrong diagnosis in spite of the very suspicious mental picture.

The course of all forms of paresis may be altered, almost always in an unforeseen manner, by two different events,—paralytic attacks and remissions.[2] The first may result in a marked, unexpected advance in all the symptoms or even in sudden death; they are by far most frequent in the demented form. On the other hand one sees abatements in the mental and nervous symptoms at almost any stage of paresis, except the very last, which delay the termination of the disease. Hoppe saw marked and long-continuing remissions in 16.8 per cent. in men and 14.9 per cent. in women; Gaupp found definite improvement in not quite 10 per cent., very pronounced in only 1 per cent. of his cases. These remissions seem to be most frequent in the agitated and expansive forms, especially the latter; one sees them less frequently and less marked in the depressive and demented forms.

[2] Halban, Jahrb. f. Psychiatrie, XXII, 358; Dobrschansky, ebenda, XXVIII, 164; Marie et Viollet, Annales medico-psychologiques, 1905, II, 102.

The quieting down sometimes occurs very suddenly, from one day to the next, although the height of the remission may be reached only gradually, perhaps after months. The patient becomes clear, sensible, orderly; the delusions recede and are referred to by the patient as dreams and imaginations. He may, himself, wonder how all that "silly stuff" came into his head. Nevertheless he, perhaps during the first few days, falls back occasionally into his old ideas and is only convinced again of their incorrectness after earnest persuasion.

The recollection of the period of mental disturbance is often confused at first, but gradually many incidents are recalled. A certain amount of insight may be developed, although many still take a wrong view of their altered behavior and declare that it was the result of external circumstances and influences. Along with this moderate clearness regarding the past there is frequently a cheerful lack of insight regarding the future. The patient feels now entirely well and is certain that he will remain so in the future; he carelessly casts aside the advice of the physician. The mood is at times one of self-satisfaction and contentment, again of depression and indifference. In the latter the patient feels tired, enervated, in need of rest and complains of all sorts of bodily ailments, especially of pressure and pain in the head. The nervous disturbances are usually more serious than the mental, still the speech and writing also may undergo marked improvement. Severe speech disorder is held by Gaupp to be a very unfavorable prognostic sign.

The condition of the patient may steadily improve so that, especially within the confines of the institution, he gives the impression of a nearly or quite normal man. From his closest relatives and friends there is hardly ever concealed a slight weakness of intelligence and memory, a blunting of his mental alertness and mood-responses as well as a certain reduction in his working capacity. Nevertheless, many such patients are able, during the

remission, to take up again such responsible vocations as railway conductors, officers, physicians. One of my patients who succumbed after he had paresis for twelve years was able, after an initial depression, not only to fill his place as telegraph operator to complete satisfaction for five years, but was promoted to higher positions, passed examinations and married; the first physical symptoms appeared two years after the relapse. Another patient who had grandiose ideas, speech disturbance, pupillary rigidity, Westphal's sign and fainting attacks, lost his delusions, took up his old position as janitor, for six years, again came down with his previous symptoms, but improved rapidly once more. With gradually diminishing mental power he was still able, save for short intermissions, to continue his work for over two years and died quite suddenly after a total duration of the disease of about nine years. Lacquer tells of a sculptor who during a remission, in spite of persistent physical signs, was able to turn out some valuable pieces.

As a rule the remissions last a few months at most; those cases in which the patients remain almost normal for longer than two to three years must be regarded as exceptions. Dobrschansky has described a case with remission lasting 14 years and a number of others collected from the literature, among which that of Halban's patient is remarkable. His paresis developed twelve years after the syphilitic infection and three years after the onset of tabes. At the conclusion of a severe phlegmon with fever lasting four months, a remission of eight years occurred, during which, however, the tabes persisted. Other cases of pronounced improvement following prolonged purulent affections have often been recorded; Steiner saw a remission after erysipelas, Marie and Pelletier after the development of a perforating ulcer.

The last stages of the disease are the same in all its forms, with the exception of those which terminate early. The patient becomes steadily duller and more demented; he no longer recog-

nizes objects or persons about him; understands neither commands nor gestures and finally is hardly more than a vegetating body in which the psychic life is almost or quite lost. At times there is a temporary excitement with monotonous cries and roars, lasting hours and days. The nervous disturbances also progress steadily. The patient is almost entirely insensitive; the weakness increases; rigidities, intention tremors, contractures and general muscular atrophy occur, so that the patient loses all capacity for voluntary movements. He grows more and more emaciated and is constantly very uncleanly, requiring to be cared for like a child. There are, to be sure, various intermediate forms leading up to this last stage of dementia and helplessness, which are differentiated from one another by varied degrees of retention of mental alertness, residuals of depressed or expansive moods and ideas, as well as by the different distribution of the various disturbances.

Termination

The termination[3] of paresis is regularly death. We are accustomed, in view of an overwhelming number of observations, to regard as paresis only those cases which end thus within a reasonable length of time. In fact, one does well to regard with greatest skepticism cases of "cured" paresis, since Nasse[4] found that of six recovered cases observed by him only one failed to have a relapse, and in this one the diagnosis was not free from doubt. Nevertheless it appears to be our duty to keep continually before us the question whether the paretic process must inevitably have a fatal outcome. There are always a small number of cases reported by reliable observers, of fully developed paresis which has undergone complete and lasting recovery. In such cases the doubt must always remain whether the disease was really paresis or one of the many other very similar cortical affections which

[3] Gaupp, Deutsche med. Wochenschr., 1904; Gaupp-Alzheimer, Centralblatt f. Nervenheilk., 1907, 696.

[4] Nasse, Allgem. Zeitschr. f. Psychiatrie, XLII, 136.

hitherto have been almost impossible to eliminate during life; there may also be disease forms of which we are entirely unfamiliar.

Of very important bearing on this whole question is the well-observed case published by Tuczek which first showed the clinical picture of paresis in 1876 at the age of 36 and again became entirely normal about the end of 1878. In 1883 tabes developed without mental symptoms and progressed gradually. In 1898 there occurred a state of excitement and confusion which resulted in mental deterioration and death 22 years after the beginning of the disease. In the opinion of Nissl who made the anatomical examination, the findings in the cortex were undoubtedly those of paresis. This observation which shows a stationary period in the paretic disease process lasting almost 20 years, reveals the possibility of a healing of the process in its early stages, especially since we repeatedly see, in this stage, remissions which, aside from the probability of a later relapse, cannot be distinguished from recoveries. Certainly we do not dare to say that in such cases there has been a complete recession of all the disease alterations. The once destroyed nervous tissue is not replaceable. Alzheimer and Schaffer have demonstrated the characteristic anatomical changes even in cases dying in good remissions. However, since we know nothing of the bearing of disturbance of certain parts to the psychic life nor of the possibility of functional representation in other parts, an interruption of the paretic process may approach the more nearly a cure, since a regeneration of such alterations which have not reached a complete disintegration is probably possible within certain limits.

In any case there can be no doubt that in paresis, although in rare instances, a very long or even a permanent arrest of the disease may occur. In fact the cases in which one is inclined to form this opinion are not so very uncommon. In every large asylum there are patients in whom a diagnosis of paresis has been

made in the beginning on account of the association of more or less typical mental disturbances with certain physical symptoms as pupillary disorder, altered reflexes, speech distortion, convulsions, but who have not progressed further toward the expected termination. In some cases this is only a prolonged remission. I recall a patient whose original diagnosis of paresis was, in the course of years, entirely forgotten, as there was a complete disappearance of all symptoms, when the paretic picture again appeared and terminated as usual in death. In other such cases the original symptoms clear up, leaving simply a more or less definite degree of dementia with or without delusions and certain unchanging residuals of physical disturbance.

As a rule, it appears that in such of those cases as are true paresis we are dealing, not with a prolonged stationary period in the process—a recovery with defect—but only with an extremely protracted course. Alzheimer had opportunity to study two cases of paresis of very long duration, one of which died in Gabersee of an intercurrent affection after a 32-year course. Paretic alterations, including disease foci of fresh appearance, were found, although these were, to be sure, extremely few. In one case the involvement of the vessels was very slight. In such cases the pleocytosis and apparently also the complement-fixation property may disappear from the spinal fluid, although Plaut obtained a positive serum-reaction in the blood and spinal fluid of a paretic of 23 years' duration, which, however, has not yet been anatomically confirmed.

A portion of these atypically terminating cases belongs, in great probability, to other varieties of cortical disease, especially luetic forms. Among 19 cases of paresis of over 8 years' duration collected by Gaupp in the Bavarian and Wurttemburg asylums by means of a circular letter, only five proved, on more careful examination, to be true paresis. Nevertheless, in the present status of the question the possibility cannot be excluded that we have

sometimes to reckon with paresis which does not progress but remains stationary at a certain stage of the disease. Brunet has recorded stationary conditions with speech-disorder, pupillary inequality, memory defect and exalted ideas lasting 20 and 30 years. Lustig has published two cases of more than 20 years, Jahrmärker and Kundt one of 9 years, Wickel one of 12 and one of 16 years' duration. Sardain has collected a number of similar observations. Here also, doubt will often arise as to diagnosis, although with the present accuracy of our clinical and anatomical methods an explanation of these atypical cases is to be expected soon. The previously-mentioned, anatomically corroborated case of Alzheimer with an exceedingly protracted course of 32 years is a striking example.

Unfortunately the exceptional cases do not alter the fact that after the experience of the various observers, nearly half of all patients with definite paretic signs die in the course of the first two years. Of 244 cases whose duration I have determined, the numbers dying in the different years of the disease are as follows:

1st	2d	3d	4th	5th	6th	7th	8th	9th	10th	14th
51	63	52	41	22	4	5	2	2	1	1

In the first year there die, according to Buchholz, 10 per cent., according to Hoppe 20 per cent. of men and 12 per cent. of women, according to Junius and Arndt 19 per cent. of men and 29 per cent. of women. Müller found 25 per cent. still alive after three years, Junius and Arndt 13.7 per cent. men and 9.9 per cent. women after four years, Heilbronner 10–13 per cent. after five years, Behr only 3.8 per cent. after six years. The average duration of the disease is estimated by Smith as 24, by Torkel as 27, by Räcke as 28, by Behr as 32 months. Junius and Arndt give 28 months for men and 24 months for women. Juvenile paresis tends to show a prolonged and irregular course. On the other hand, according to the authors mentioned, the course is shorter as the age is greater. It sinks for example from 31.9 months in

men and 28.4 months in women between the 30th and 35th year to 25.4 and 18.2 months respectively between the 55th and 60th year. In constitutionally defective persons paresis has a longer course, while association with tabes, according to the opinion of most observers, does not prolong the duration.

The fatal outcome of paresis is brought about by a great variety of causes; according to Obersteiner it occurs more frequently in winter, possibly on account of the patient's defective temperature regulative mechanism. Aside from the occasionally successful attempts at suicide during the early periods, life may come to a sudden and unexpected ending in paretic attacks at any part of the course. In the last stages of the disease aspiration pneumonias (saliva and food), especially during attacks, are by far the most common cause of death. After Hoppe's figures 32.8 per cent. of men and 18.5 per cent. of women die during paretic seizures. Septicemia and fat embolism also come into consideration, as they may develop from bed-sores or bladder inflammation (pyelitis), resulting from the restlessness and insensibility of the patient. A few patients die from suffocation during vomiting or in stuffing their mouths with food, especially bread, and aspirating a portion into the larynx. Finally, the so-to-speak natural termination which we observe in those cases which escape the dangers described, is a severe marasmus with death from cardiac failure. The patients emaciate to mere skeletons; the whole musculature undergoes extreme atrophy; the temperature sinks, often for long periods, below normal; the pulse is slow and grows weaker and finally indiscernible until at last life becomes entirely extinct.

CHAPTER III

Post-Mortem Findings

The pathological anatomy of paresis reveals a number of alterations in the central nervous system which in their totality may be regarded as diagnostic of the disease.[5] The occasionally found hyperostoses and exostoses of the skull are not to be considered as important and are not at all rare in the normal and in other mental diseases. Marked osteoporosis is common and is apt to be more marked in other bones, especially the ribs.

The changes in the meninges are more important. The dura is often adherent in places to the calvarium, less often universally; sometimes it can be separated from the bone without injury. It is not uncommon to find pachymeningitis interna and hematoma of the dura, usually as a mere delicate film but sometimes in the form of a thick coat made up of several layers, or of an extensive fresh extravasation of blood, generally over the vertex. One also frequently sees more or less widespread superficial hemorrhages. The pia, as shown in Fig. 19, is always more or less cloudy and thickened, sometimes excessively, especially along the vessels. Microscopic examination shows an increase in connective tissue and localized infiltrations with lymphocytes and plasma cells as well as occasional mast-cells and gitter-cells; a similar condition exists in the choroid plexus. These alterations are generally most apparent over the anterior and middle portions of the hemisphere convexities, also on the inner surfaces, but less prominent on the basal surface and entirely lacking over the occipital lobes.

[5] Nissl, Histologische und histopathologische Arbeiten, Bd. I, 1904; Cramer, Handbuch der pathol. Anatomie des Nervensystems von Flatau-Jacobsohn-Minor, 1470, 1903; Alzheimer, Histologische Studien zur Differentialdiagnose der progressiven Paralyse. Habilitationsschrift, 1904.

Alzheimer found this distribution in 142 cases out of 170. Sometimes the thickening is more marked on one side, as is the case in the illustration; in this brain it is also excessive over the temporal regions. Occasionally bony plates are found in the pia. The veins are greatly distended, especially in galloping paresis,

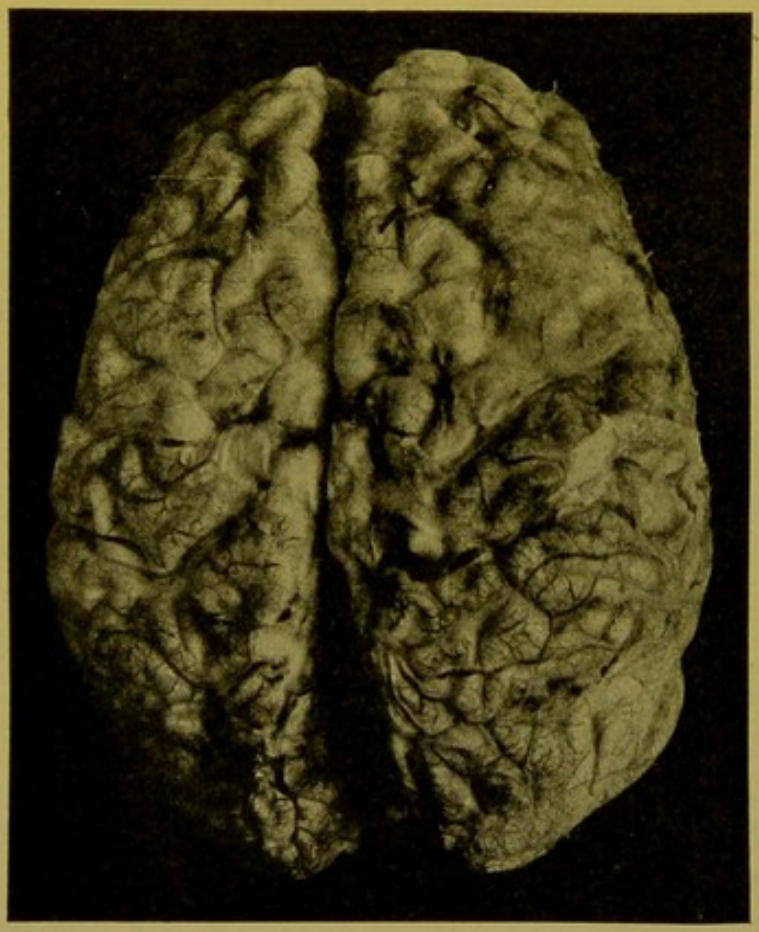


Fig. 19. Paretic Brain.

and often show thickened walls; the Pacchionian granulations are frequently over-developed.

The brain in old-standing cases is always atrophic. The convolutions are reduced, especially in the anterior parts; local sinking-in of the brain substance occurs, over which the pia is stretched in the form of serum-containing cysts. The cortex is

also reduced and, especially in the frontal lobes, is so adherent to the pia in places that this cannot be removed without tearing the substance beneath. This is especially true in late autopsies. The atrophy involves by preference the frontal lobes, which show the greatest involvement in about 80 per cent. of the cases according to Alzheimer. Exceptionally, there is pronounced atrophy also in the temporal lobes or central gyri, less often in the parietal lobules or occipital lobes. Now and then the whole cortex is diffusely atrophic. The ventricles are more or less widely dilated, their ependyma, especially that of the fourth, almost always shows numerous, prominent granulations. Weigert's studies have shown these to consist of hyperplasia and hyaline degeneration of the neuroglia with loss of the epithelial covering. The amount of fluid appears, as a result of the atrophy, to be increased both in the subdural space and in the ventricles (hydrocephalus externus and internus).

The weight of the brain was found by Ilberg[6] to show an average reduction of 152 grams, a loss which is borne almost entirely by the cerebral hemispheres; there is often a decided difference in the two sides. Brunet reached similar results and found a disparity of 50–60 grams mostly in favor of the right hemisphere. Alzheimer once found a difference of 155 grams. The loss of weight is greatest in the frontal lobes according to Boumann. The average weight of the brain in paretic men is 1,236 grams, after Ilberg, with variations between 910 and 1,549 grams; in women, 1,089 grams with limits of 802 and 1,305 grams. Now and then one encounters an unusually heavy brain, but there must remain a doubt whether these are cases of true paresis and not some other form of cortical disease with excessive glia-development. In the chemical study of the brain-mass Barratt found an increase in amount of water and a decrease in the content of phosphorus. In occasional cases a gumma is found in

[6] Ilberg, Arch. f. Psychiatrie, XXXI, 936; Brunet, Annales medico-psychol., VIII, 11, 251, 1900.

some part of the brain, also very rarely, some other kind of tumor, a glioma or a spindle-cell sarcoma, in whose neighborhood the paretic process may be especially marked.

The *microscopical examination* shows outspoken changes first of all in the cortex.[7] In the cortical nerve-cells one finds a great number of more or less serious alterations, as were described by Nissl and others at an earlier date. Further study has shown that pictures here referred to are not characteristic of the disease-process, but, so far as we can now determine, constitute the general expression of the injuries suffered by the cell during life. We find the same pictures again among a great variety of pathological conditions. Many of these affections, as, for instance, the acute swelling, are apparently capable of recovery. In others, such as the very frequent and early chronic cell-sclerosis, the cell may persist for a long time although in an altered form, perhaps even dead; not infrequently one can see in the diseased and shrunken cells the traces of recent, acute exacerbations of the disease-process. In contrast to this, we have Nissl's so-called grave alteration which probably results inevitably in the destruction of the cell; it is recognized by the lysis of the stainable substance with reduction in size and rounding out of the limiting membrane, loss of the stroma and the bluish-violet stain taken by the nucleus. Even the acute disease and cell-sclerosis can without doubt lead to destruction of the affected cell.

The alterations spoken of, with the exception of the acute, never affect the whole cortex at once. On the contrary, many local differences are found in the extent and severity of the de-

[7] Binswanger, Die pathologische Histologie der Grosshirnrinden-Erkrankung bei der allgemeinen progressiven Paralyse, 1893; Nissl, Arch. f. Psychiatrie, XXVIII, 989; Heilbronner, Allgem. Zeitschr. f. Psychiatrie, LIII, 172; Ris, Correspondenzblatt f. Schweizer Ärtze, XXXVII, 7 u. 8; Lawrence, American Journal of Nerv. and Mental Diseases, 1903, 533; Evensen, Det anatomiske grundlag for den paralytiske Demens. Habilitationsschrift, 1905.

structive process. Thus the occipital cortex and particularly the region of the calcarine fissure, as well as the central convolutions, especially the anterior, are relatively little affected, while the frontal, parietal and many times the temporal lobes are greatly involved. No absolute rule, however, applies in this regard. In a very acute course of the disease, on the one hand, there is a tolerable uniformity of the process in the whole cortex while, on the other hand, in the far more numerous chronic cases there is a great diversity of distribution of the alterations in the various lobes and convolutions, in spite of the common characteristics. Even in the same spot in the cortex one may see various grades of the pathological alterations lying side by side with numerous cells which appear entirely normal. Only in a very severe, acute course or after a long duration of the disease does one see all the cells of the cortex showing in various degrees indications of the disease. In all cases a greater or lesser number of cells is completely destroyed. In a markedly atrophic cortex a dropping out of cells to a certain extent by layers is to be detected. The third layer is especially prone to severe affection, while the second appears to be better preserved, because in it the sclerotic change which tends less to destruction of cells, is the more prominent. In the severest cases of cortical devastation, practically the whole of the nervous tissue finally succumbs so that only a few scattered, sclerotic cells are found remaining in the cortex.

The destruction of nerve-fibers is in direct ratio to that of the nerve-cells. Tuczek[8] has studied these in detail by the finer methods (Exner's, Weigert's). His findings showed that in all long-continuing cases of paresis a very widespread loss of fibers is present, so that in the last stages it is difficult to demonstrate any fibers at all in the cortex. The fine supra-radial network of the second and third layers seems to be the first to go, then the tangential fibers of the most external layer, coursing parallel to

[8] Tuczek, Beiträge zur pathologischen Anatomie und zur Pathologie der Dementia Paralytica, 1884.

the surface, and finally also the radiating fibers, while, at the same time, the white matter is reduced. Figs. 20 and 21 may give an idea of this alteration; in these the fiber network of a normal and

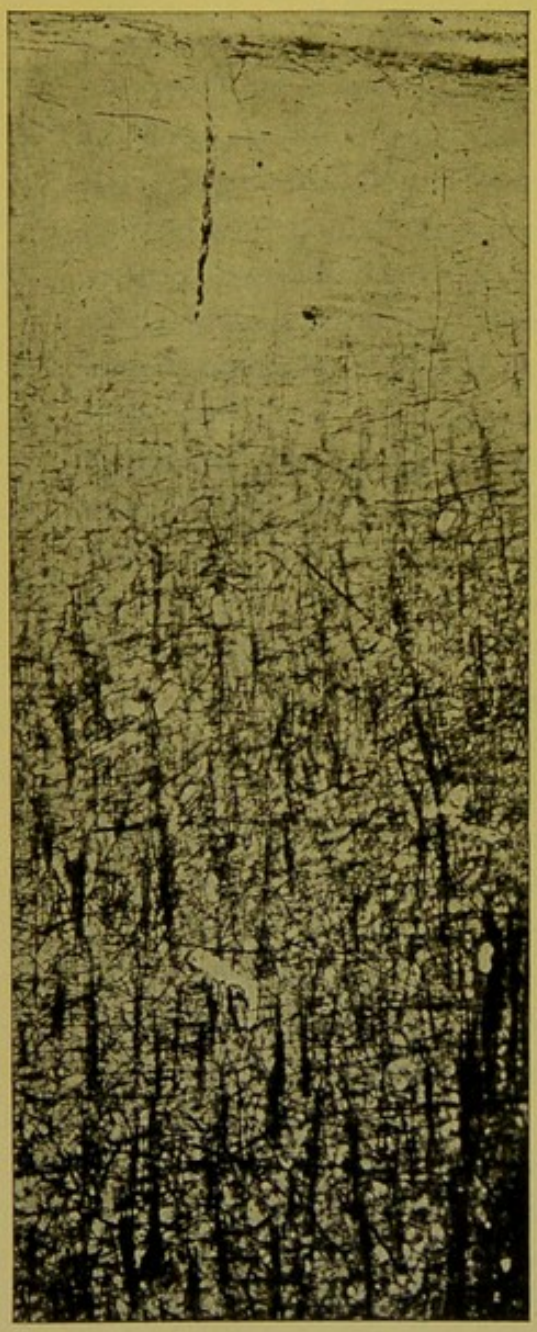


Fig. 20. Nerve-fibers in Cortex of Healthy Brain.

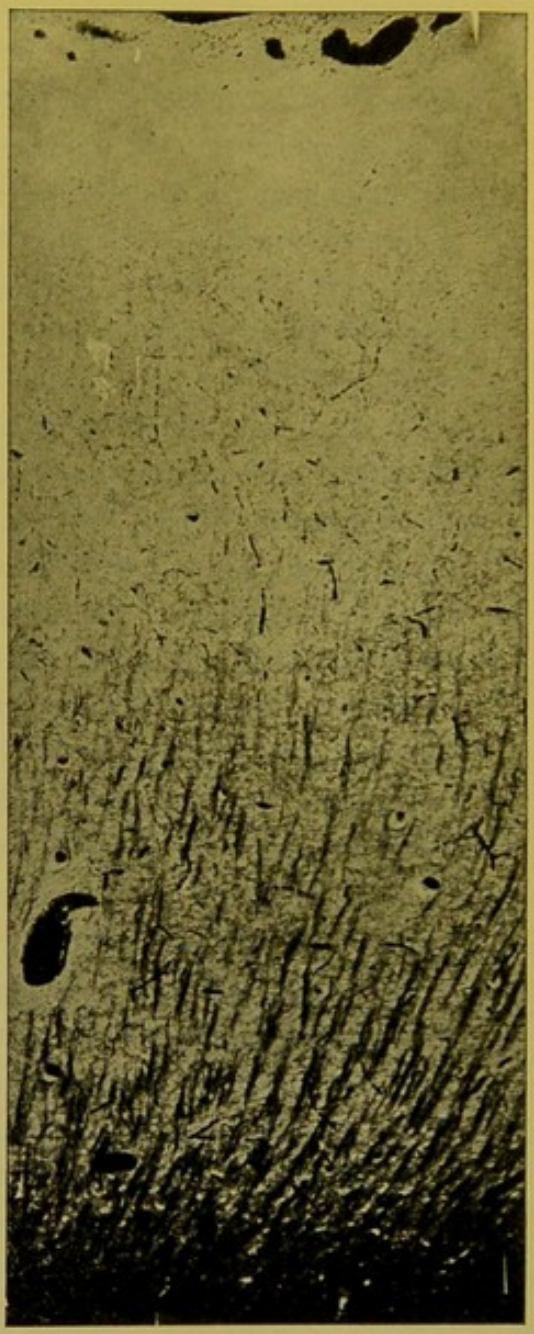


Fig. 21. Nerve-fibers in Cortex of Paretic Brain.

of a paretic brain are shown. One sees that in the latter the tangential fibers and the horizontal fibers of the cortex are practically entirely gone, while the more resistant radial network shows only a very decided thinning out.

Fischer has described in addition, as a frequent finding in paresis (65 per cent. of the cases), a loss of fibers in spots in the deeper cortical layers and extending also into the marrow, which involves the central convolutions more commonly, less often the frontal lobes, and appears to be connected with the vessels. The axis-cylinders are preserved in the foci; in their neighborhood there is slight glia overgrowth. In other respects, the studies of Schaffer have shown that the fiber degeneration has the same distribution as the cell alterations; it is most marked in the frontal portion and on the basal surface, also in the parietal lobules, in the superior temporal gyri and in the lower portion of the island. It is, in itself, just as little characteristic of paresis as the cell changes, and is present in a large variety of other cortical affections, especially the senile and epileptic. To be sure, the frequency, the extent and the severity of the alterations in the nervous tissue are likely to be in general much greater in paresis than in any other cortical disease.

The condition of the fibrils in the paretic cortex has been studied by Marinesco, Brodmann,[9] Sciuti and others. Swelling, massing together, waviness, beading and crumbling of the fibrils, dissolution into rows of granules and complete disappearance were found. The alteration seems to be most prominent in the peripheral network, next in the cells themselves and to begin usually about the nucleus and at the base while the dendrites and axis-cylinder processes resist longer. In sclerosed cells nearly all fibers are destroyed. The layer of small and middle pyramidal cells is usually most affected. The distribution of the disturbance is similar to that of the cell-changes previously described but cells

[9] Brodmann, Journal f. Psychologie u. Neurologie, V, 186; Sciuti, Annali di neurologia, XXV, 6.

which are nearly or completely normal are found alongside of those which are severely affected. The same disturbance in the fibrils occurs also outside the cell. The finer fibers, especially, disappear in large numbers, mostly in the cortical layers. On the other hand the fibrils may persist even when the myelin sheath is affected.

Very severe and extensive alterations are suffered by the ectodermal tissue of the cortex, the neuroglia. We have learned chiefly through the classical researches of Weigert[10] that every loss of nervous tissue is accompanied by a hyperplasia of the surrounding neuroglia. We see therefore in paresis an exuberant growth of the connective-tissue element corresponding to the

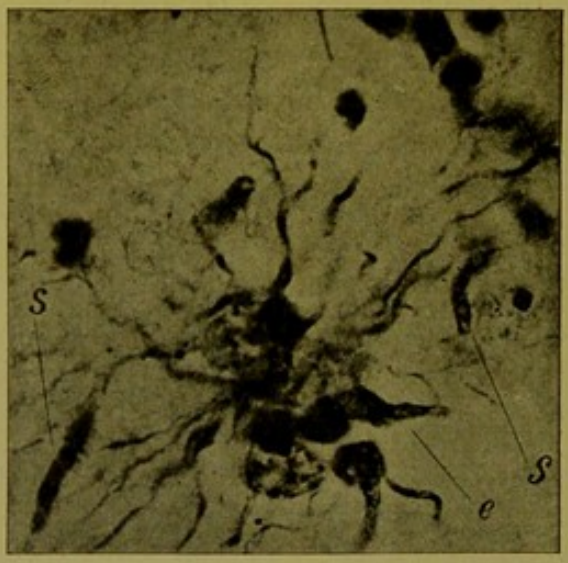


FIG. 22. Spider-cell in Paresis.

wiping out of many cells and fibers. As an indication of this there are apt to be swelling of the protoplasm and increase of chromatin in the neuroglia cells, with kinetic figures in the nucleus. There are, on the one hand, a noticeable increase in the number of glia cells, whose nuclei are very prominent in the paretic cortex, and, on the other hand, an abundance of neuroglia fibers. In this

[10] C. Weigert, Beiträge zur Kenntnis der normalen Menschlichen Neuroglia, 1895.

process a prominent rôle is played by the so-called astrocytes or spider-cells which have long been recognized and sometimes develop very fantastic shapes; they occur chiefly about the vessels and in the deeper layers of the cortex. As Weigert has pointed out, these serve as supporting points for numerous fibers which are formed by and belong to them. An example is shown in Fig. 22. Here we see a clump of light and dark glia nuclei with serpentine fibers in a star-shaped arrangement about them.

The new-formation of glia fibers is greatest at the surface of the nervous tissue, in other words in the cell-free border of the cortex. Fig. 23 shows a normal amount of glia at the cortical

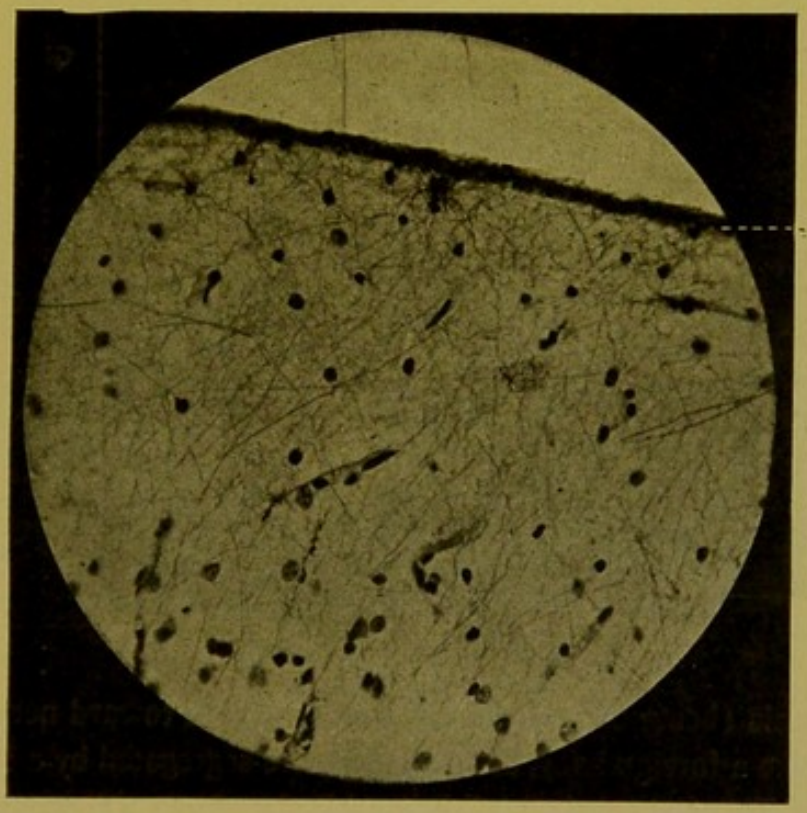


Fig. 23. Normal Glia. (Gl.)

surface, while in Fig. 24 we see the marked widening and thickening of this fiber network in paresis; at the same time the increase in glia nuclei and fibers in the cortex is apparent. Still

more marked is the increase in glia fibers in the section from the cerebellum shown in Fig. 25. Here we see that the glia has not only formed an extremely thick network and broad envelope in the cortex itself but it has grown out in the form of brush-like processes into the infiltrated pia and thus formed an adhesion between this and the cortex.

The free cortical surface is not the only limit of the nervous tissue. Nissl, in agreement with Weigert, has emphasized that

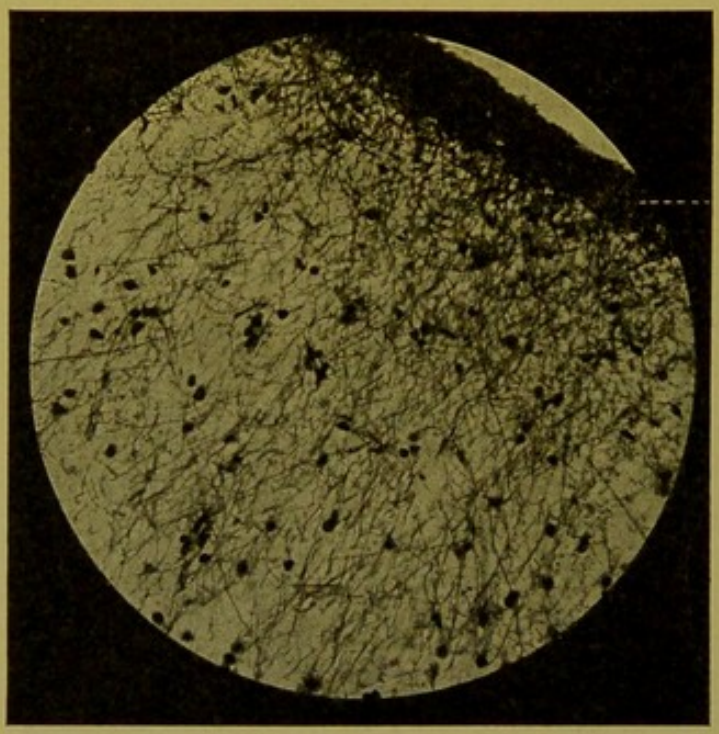


Fig. 24. Glia in Paresis (Gl).

the vessels (being of mesodermal origin) behave toward nervous tissue like a foreign body, and therefore are segregated by a layer of glia. This glia envelope of the vessels also shows a pronounced thickening in paresis, so that in advanced cases, according to Alzheimer's article, almost all of the glia of the deeper layers seems to lend itself to the formation of sheaths for the vessels. The glia cells send especially large fibers toward the vessel in order to unite with its glia sheath. One recognizes these relations in Fig.

26. Also in Fig. 22, *e* indicates an endothelial cell of a capillary to which the spider-cell is attached.

The hyperplasia of the glia stands only in general, not in particular relation to the destruction of the nerve-cells. On the one hand we observe widespread loss of cells without remarkable increase of glia; on the other hand we often find nearly or entirely normal cells in the midst of marked glia overgrowth. It therefore follows that the destruction of the cells is quite independent

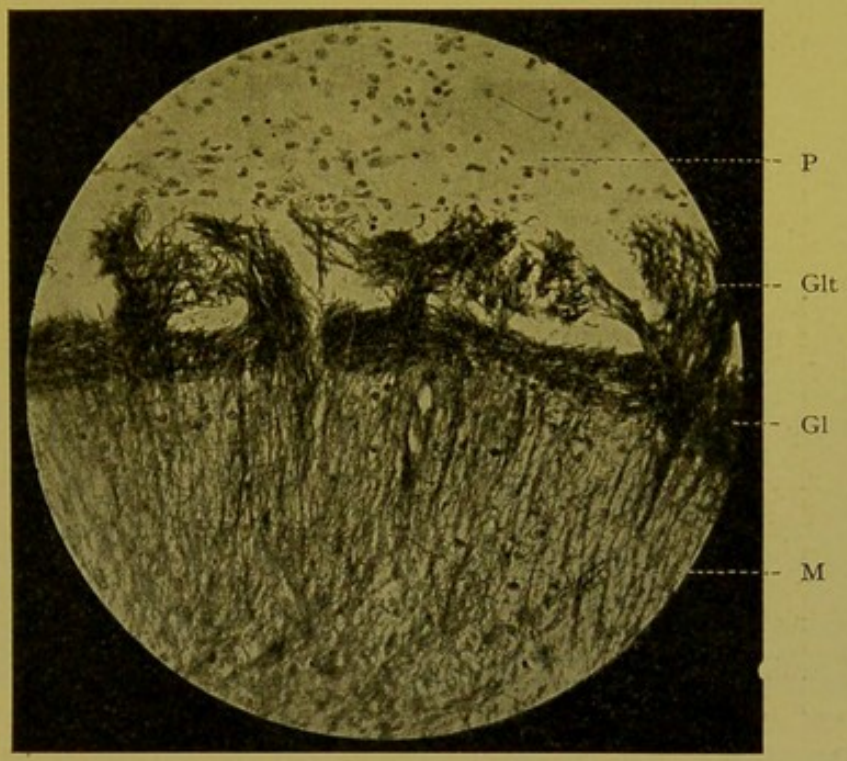


FIG. 25. Brush-like Proliferation of Glia. P. = Pia; Glt. = Glia Tufts; Gl. = Superficial Glia Layer; M. = Molecular Layer of Cerebellum.

of the glia hyperplasia and not at all conditional upon the latter. The nerve tissue is damaged directly by the disease process; the glia overgrowth is a common, although sometimes a mixed or late-appearing accompaniment. Besides the numerous proliferative processes in the neuroglia tissue, there are also many degenerative changes, sclerosis, pigmentary degeneration, vacuo-

lization, swelling and breaking up of the nucleus. The satellite cells clinging to the nerve cells show later an indication of de-

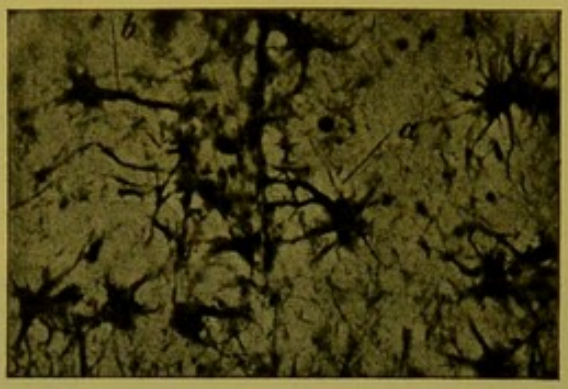


FIG. 26. Vessels with Spider Cells in Paresis.

generation, also the smallness and dark staining of the nucleus, which may terminate in complete destruction.

The mesodermal elements of the cortex, the vessels, are also profoundly affected by the paretic process. First there is endothelial proliferation with nuclear division. Then there is regularly and especially in cases with frequent paralytic attacks, a new-formation of vessels by branching which sometimes reaches an enormous extent; endothelial cells grow out into the surrounding tissue, are accompanied by adventitial cells and form new capillary loops. This is very prominent in Fig. 27 and we see very numerous and much widened capillaries with frequent peculiar tufts. The vessels of the cortex are sometimes so increased that one can see their lumens in a cut surface with the naked eye. One observes in addition a thickening of the vessel wall by endothelial placques in the wider openings of the larger vessels leading into the cortex from the pia. There occurs a new-formation of elastic tissue and, in old cases, occasional hyaline degeneration[11] of the wall. The adventitia shows pronounced increase, constituting further evidence of degenerative change.

[11] Alzheimer, Arch. f. Psychiatrie, XXX, 18.

Occasionally there is narrowing, less often small aneurisms and capillary hemorrhages.

The particularly characteristic alteration in the vessels, however, is the accumulation of cells in the widened adventitial lymph-

FIG. 27. Vessel Increase in Paresis.
Vl. = Vessel-Loops; Vsp. = Sprouting Vessels.

space, which is only found, in such form and so widely distributed, in paresis. The cells are never leucocytes (which only occur with associated infection), but are partly lymphocytes, partly peculiar cells first described in this connection by Alzheimer and regarded by Nissl as plasma-cells in the sense of Marschalko. They consist of a protoplasm body which is usually round but often long or angular and is irregularly stained with commonly a light area

near the center. The nucleus generally lies not exactly in the center and has about its margin a number of very dense, deeply staining chromatin granules and one or two nuclear bodies. In the opinion of Unna and Verrati these cells come from the adventitial connective tissue, while Marschalko and Nissl hold them to be blood cells which have wandered into the lymph-sheath. Their peculiar construction and their location in the lymph-spaces are shown in Fig. 28. Here one sees plasma cells, which may be

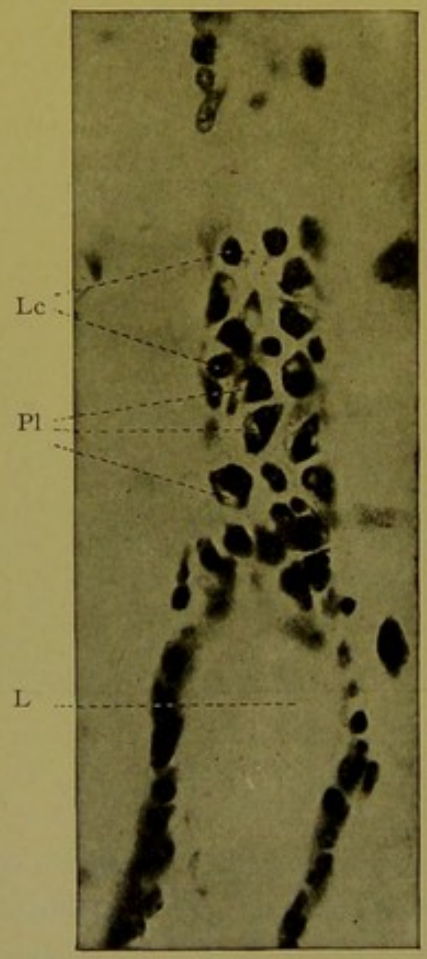


FIG. 28. Plasma Cells in Sheath of Vessel in Paresis.
Lc. = Lymphocyte; Pl. = Plasma Cell; L. = Lumen.

recognized by their light area, arranged like a cloak about the vessel and shown in the cross section to lie in the adventitial

sheath. Now and then a plasma cell is seen in the cortical substance, but always in the neighborhood of a vessel.

Vogt,[12] under the direction of Nissl, first demonstrated the close relationship between plasma-cell infiltration and the paretic disease-process. He found this picture in large numbers in the 14 cases of paresis studied by him, while it was entirely lacking in 23 mentally sound cases and 40 cases of mental disease other than paresis. In one of the latter a few plasma-cells were found but only a single case, an epileptic idiot in whom the possibility of juvenile paresis was, at least, not excluded, showed a picture like that of paresis. Continuation of the studies from various parts has shown that plasma cell infiltration never occurs in the healthy cortex, but may be found in a number of other diseases than paresis; most important, in syphilitic meningo-encephalitis, also in the non-purulent form of encephalitis, in carcinomatous and tuberculous affections, in rabies, in sleeping-sickness and in the neighborhood of focal softenings. On the other hand not a single case of paresis has been reported in which it failed; Alzheimer found it in all the 170 cases observed by him. It is most prominently developed in the acute stages of the disease. Later in the course the plasma cells show signs of degeneration, a tendency to pigmentation and vacuolization. Thus, in very protracted cases, the infiltrate may gradually disappear almost entirely and only persist in occasional scattered foci of fresh disease.

It must be stated, however, that the general arrangement and distribution of the plasma-cell infiltration shows certain peculiarities which distinguish it from the other disorders mentioned. In the first place the alteration is not confined to circumscribed regions or layers, as is commonly the case in other affections, but is widely distributed; its occurrence usually coincides in general, as Vogt mentioned, with the distribution of the paretic

[12] Vogt, Monatsschr. f. Psychiatrie, IX, 211; Mahaim, Bull. de l'académie royale de médecine de Belgique, 27. Juli, 1901; 27. Dez., 1902.

process. It is most marked by far in the frontal cortex while in the occipital region it is weaker and may be absent entirely. It is worthy of mention that a clearer knowledge of the localization of the cell infiltration as well as of the manner of its occurrence and disappearance will afford a means of differentiating those diseases in which to-day this is impossible. The complete anatomical picture, at least, will give us help. The diseases which come most into consideration, however, will, with the exception of the syphilitic forms, offer no difficulty on clinical grounds.

A further peculiar cell type, whose presence seems to bear a close relationship to the paretic process, are the rod-cells recently described by Nissl. They are elongated, very small, sometimes crooked cells consisting almost solely of a light nucleus with a single nucleolus overreached more or less by the cell body at both ends. They lie usually in the neighborhood of the vessels, arranged parallel to the long axis of the nerve cells, but in the deeper layers more irregularly placed. Nissl and Alzheimer regard them as off-shoots of the vessel-walls, while Sträussler and Cerletti defend their origin from the neuroglia. Rod cells are found also in other cortical affections but generally only in small numbers and not remote from the meninges as in paresis. Spielmeyer found them numerous only in sleeping-sickness. The very elongated forms also seem to be more characteristic of paresis. In Fig. 22 rod cells are seen in S., one of which is bent at an angle.

The cortical picture of paresis is composed of the sum of the alterations which have been described, as shown in Figs. 30–33. Fig. 29, which is from a healthy brain, will serve for comparison. The first two pictures illustrate fresh processes, while the next two are of advanced cortical degeneration. In Fig. 30 the greatly enlarged and infiltrated vessels are the most noticeable feature. One notices, so far as is possible in the low magnification, the sharp, concave margins and deep staining of the processes of the

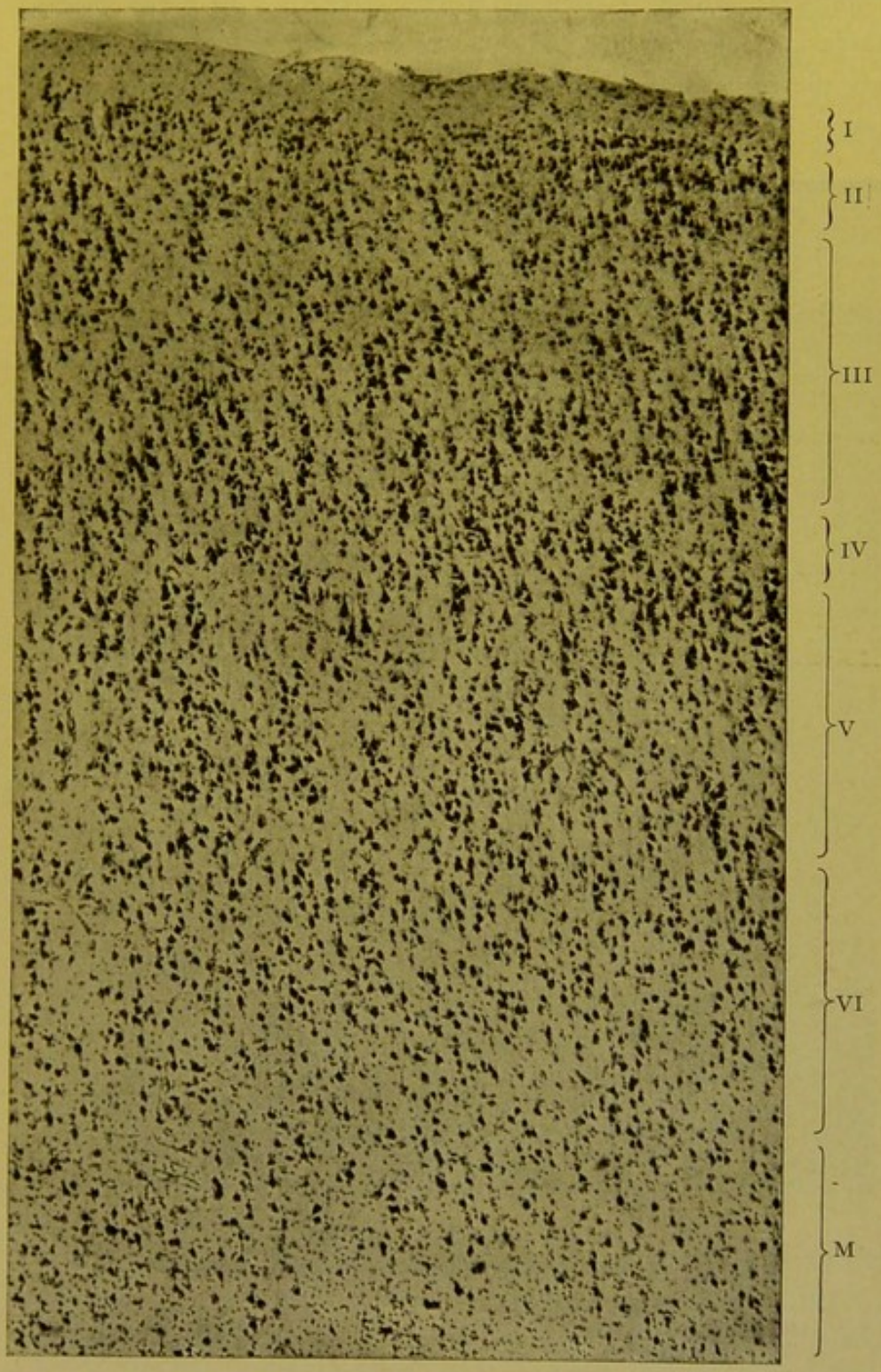


FIG. 29. Healthy Frontal Cortex.

nerve-cells which is peculiar to cell sclerosis. In many places there are spaces which indicate the dropping out of cells; these are the more prominent since the cells in general appear rather more closely arranged than in health, on account of the loss of

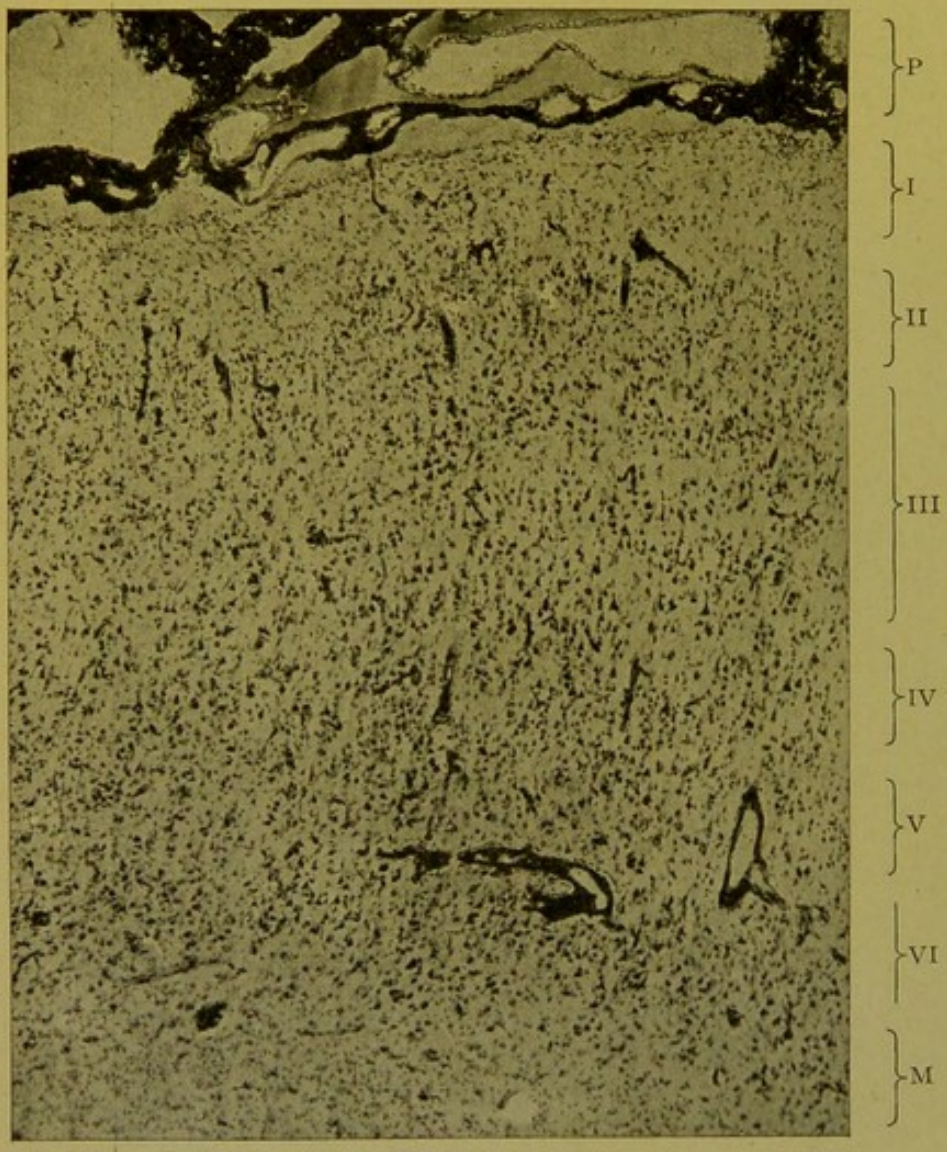


Fig. 30. Frontal Cortex in Paresis.
P. = Pia; I.–VI. = Cortical Layers; M. = Marrow.

cortical substance between them. The arrangement of the cells into regular vertical rows, as seen in the picture of normal cortex,

is not a little disturbed. They often lie obliquely and confusedly.

The distortion is less marked in Fig. 31. Here, nevertheless, the cells appear concave, closely packed. Between them, especially in the second layer, lie numerous glia nuclei. The peripheral layer appears widened. The vessels are dilated. In the

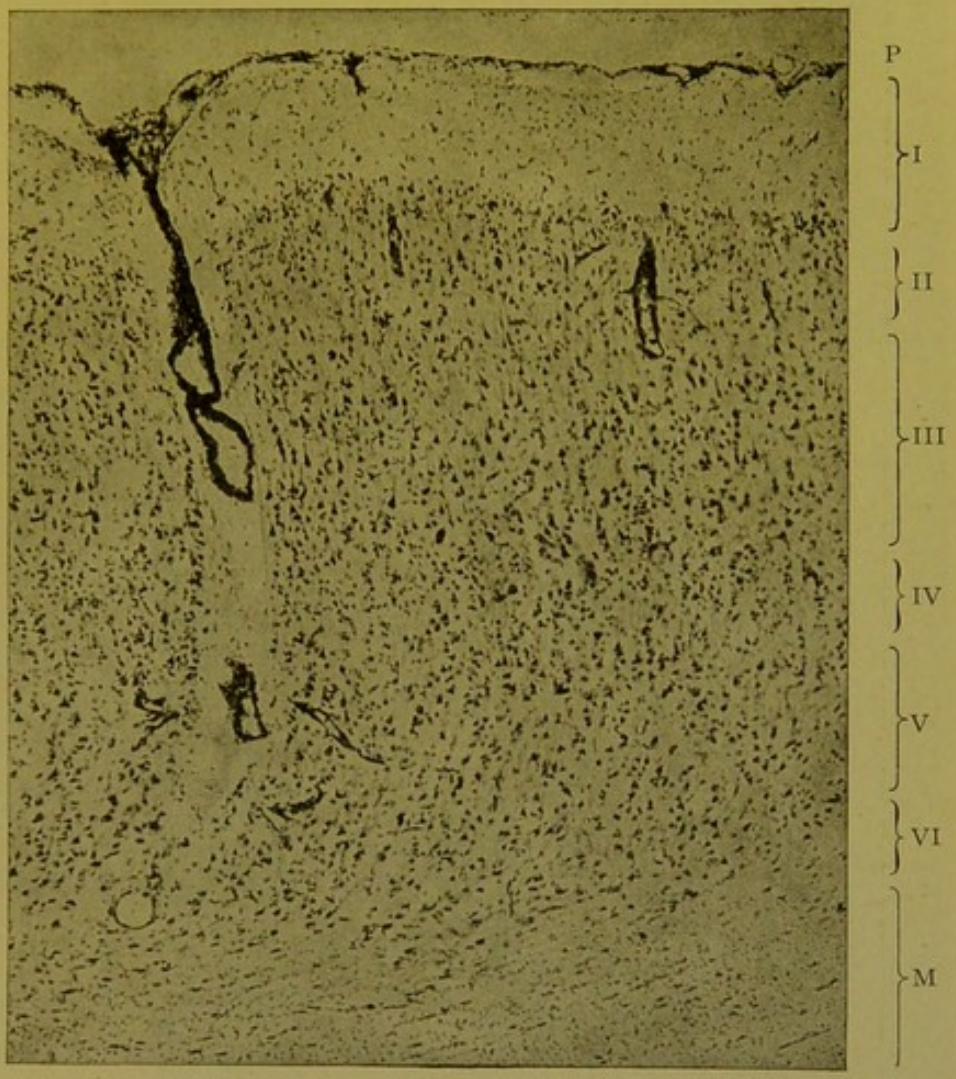


Fig. 31. Frontal Cortex in Paresis.

neighborhood of the large, excessively infiltrated vessels leading in from the pia, all the nerve cells have disappeared. In Fig. 32, besides the very pronounced loss of cells, of which only a very

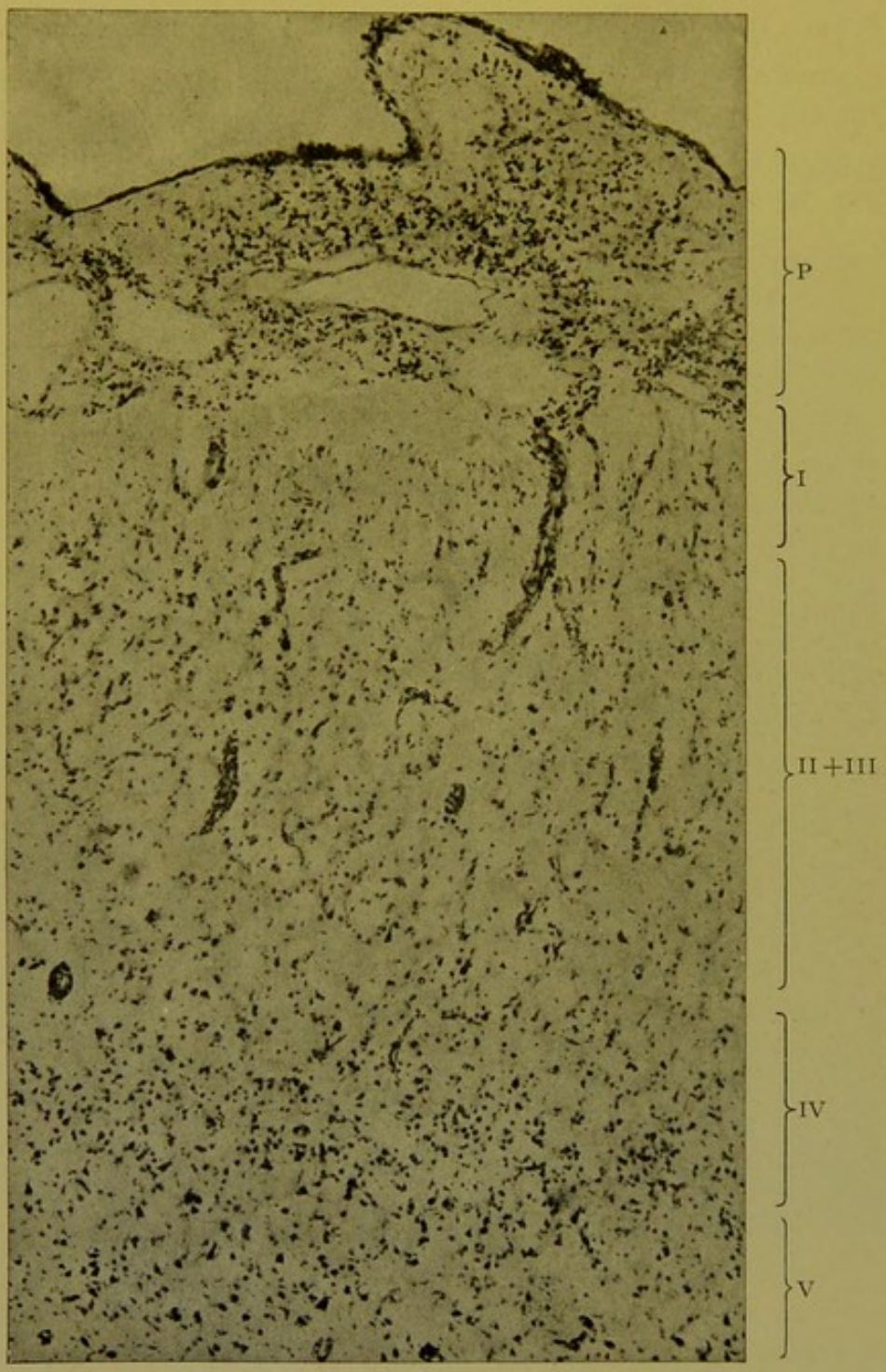


FIG. 32. Extreme Devastation of Cortex in Paresis.

few can still be seen, and the increase of glia nuclei, especially in the deeper layers, there is complete destruction of the cortical architecture. The pia is greatly infiltrated and is adherent to the cortex over a large area. An extreme destruction of nervous tissue, finally, is shown in Fig. 33. In the third and fourth layers almost no cells are visible. The whole cortex has shrunken into a narrow strip, while the peripheral layer in which are numerous glia nuclei is not reduced. The infiltration is no longer prominent in vessels or pia.

The last picture shows plainly that in paresis an atrophy of the cortex from dropping out of nervous tissue can be so excessive that the breadth is reduced sometimes one half. Some places, especially about the vessels, remind one of cicatrices. The above

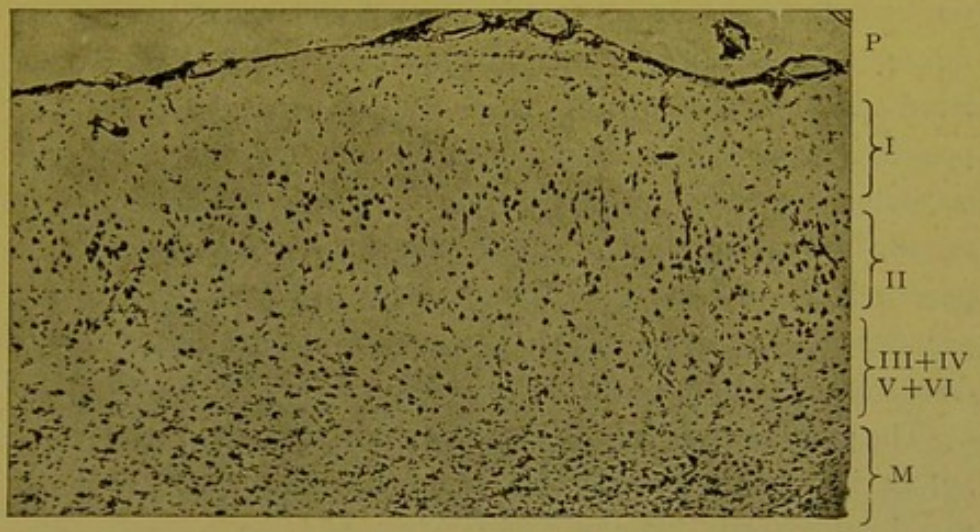


FIG. 33. Shrunken Cortex in Paresis.

described distortion of the cortical picture exists from the beginning. The nerve-cells do not stand in regular rows but appear wind-blown. In many places they are crowded together; in other places there are large areas filled only by glia-tissue and vessels. Since in a long protracted case the cells are often closely arranged so that they sometimes appear more numerous than normal, one can hardly doubt that there must have been a loss of tissue be-

tween them. Besides the loss of myelinated fibers a far-reaching disturbance must be thought of in the gray network between the cells, of whose extent and importance we perhaps still have all too incomplete a conception.

If we come now to consider the question how far the cortical changes described are characteristic of paresis we must admit that any one of the alterations may be observed also in other forms of disease. Nevertheless, when taken collectively, especially if found in various parts of the cortex, they constitute as safe evidence of this particular disease as does the constellation of clinical symptoms any of which alone is insufficient to establish paresis. Of all the changes which have been described, the accumulation of plasma-cells in the vessel sheaths seems to be of most use in the recognition of paresis, since it is never absent in that disease and generally shows also a characteristic distribution. Further, the occurrence of numerous rod-cells, the endothelial proliferation and new formation of vessels, the wide extent of severe cell-alteration and the increase of glia in the deeper layers require special mention. Important also for the diagnosis of paresis is the distribution of the changes in pia and cortex, the pronounced involvement of the frontal portion, the relative freedom from alteration of the occipital lobes.

The peculiar changes confined to certain large cortical regions, but having a very general distribution in these, particularly the vessel infiltration, the glia proliferation and the rod-cells, distinguish the anatomical picture in paresis from the changes in syphilitic meningo-encephalitis and tubercular meningitis, since in the latter, the process is directly dependent upon the meningeal involvement. The shrinking and distortion of the cortical picture may be regarded, to a certain extent, as diagnostic of paresis. The nerve tissue of the cortex is destroyed to a much greater degree than in any other disease and there is at the same time an extensive vessel-increase which may play an essential rôle in the dis-

turbance of the cell arrangement, especially since it may show itself at a time when the loss of nervous tissue is still relatively slight. In idiocy, in dementia præcox, in senile dementia, where also numerous cells and fibers go to pieces, the general structure of the cortex remains intact; one sees gaps in the cell layers filled by glia, but without the arrangement being otherwise disturbed. Only in severe arteriosclerosis can a similar picture occur.

Besides the finer alterations we frequently see in the cortex small softened foci which reveal themselves by slight detachment of the superficial cortical layer or of the whole cortex from the white matter. Gross changes which one might assume as explanations of paralytic attacks, especially hemorrhages, are, however, very rare; in Nissl's opinion they are probably always the result of septic disease. On the other hand Lissauer, Starlinger[13] and others have offered proof that the focal symptoms observed during life, the local paralyses, hemianopsia, word-deafness, mind-blindness and aphasia, correspond to definite circumscribed areas of greater severity of the process in the cortex; furthermore that secondary degeneration can be traced from these foci far into the white matter, the basal ganglia, the crura and even into the spinal cord. Finally, it is to be mentioned that besides the paretic findings, syphilitic changes can often be discovered in some part of the cortex. Aside from the easily recognized presence of large gummata, there may be either scattered small gummatous nodules along the cerebral vessels or endarteritic disease.

Alterations similar to those in the cortex are found widely distributed in other parts of the brain, as may be shown by a glance at the very marked loss of weight. The fiber-mass of the hemispheres shows, as a rule, scattered degenerations which only occasionally leave a whole bundle unaffected. Less common are foci of degeneration or tract degeneration of definite conduction paths from circumscribed disturbances in the cortex. In the large

[13] Starlinger, Monatsschr. f. Psychiatrie, VII, 1; Storch, ebenda, IX, 401.

basal ganglia, in the central gray matter and even in the cerebellum extensive fiber-degeneration is demonstrable. Lissauer saw, after especially severe involvement in certain parts of the parietal and occipital cortex, corresponding circumscribed fiber degeneration in the optic thalamus, a finding which was corroborated by Alzheimer. Räcke[14] has demonstrated alterations in the pulvinar, in the internal geniculate, in the middle gray of the anterior colliculus, in the formatio reticularis, in the olive, the pontine nuclei and in the nuclei of the floor of the fourth ventricle. Similar findings in various regions of the brain-stem, especially in the medulla and the hypoglossus nucleus, have been made by a number of other observers. In the central gray matter small hemorrhages sometimes occur (association with alcoholism).

The alterations in the cerebellum are in general of the same sort as those in the cerebrum, but less severe. Some of the cerebellar convolutions appear shrunken and show cicatricial depressions. In the pia there is the same process, proliferation of the vessels, infiltration with lymphocytes, plasma-cells and rod-cells, adhesions. Especially marked and severe is the degeneration of the granules in the granular layer which goes along with destruction of fibrils and strong glia proliferation; even in the molecular layer the glia is much increased. In the Purkinje cells Moriyasu saw the flask-like fibril network and the processes destroyed. Many cells disappear entirely. Anglade and Latreille describe also islands of sclerosis. Sträussler states that the amygdalae and the under surface of the cerebellum usually seem to show the greatest involvement. Fig. 34 gives a picture of a paretic cerebellar convolution which may be compared with Fig. 35, showing a normal cerebellar cortex. One notices at once the shrinkage of the whole convolution, which involves especially the granular layer; the larger part of the granules have disap-

[14] Räcke, Arch. f. Psychiatrie, XXXIV, 523; Sträusler, Jahrb. f. Psychiatrie, XXVII, 7.

peared. The molecular layer is also somewhat reduced; it contains numerous closely arranged glia nuclei. Of the Purkinje cells there are only a few degenerated remains at the base of the convolution.

In the spinal cord[15] one observes occasional pachymeningitis and frequent leptomeningitis, more marked over the dorsal columns. By far the most frequent degeneration is found in the pos-

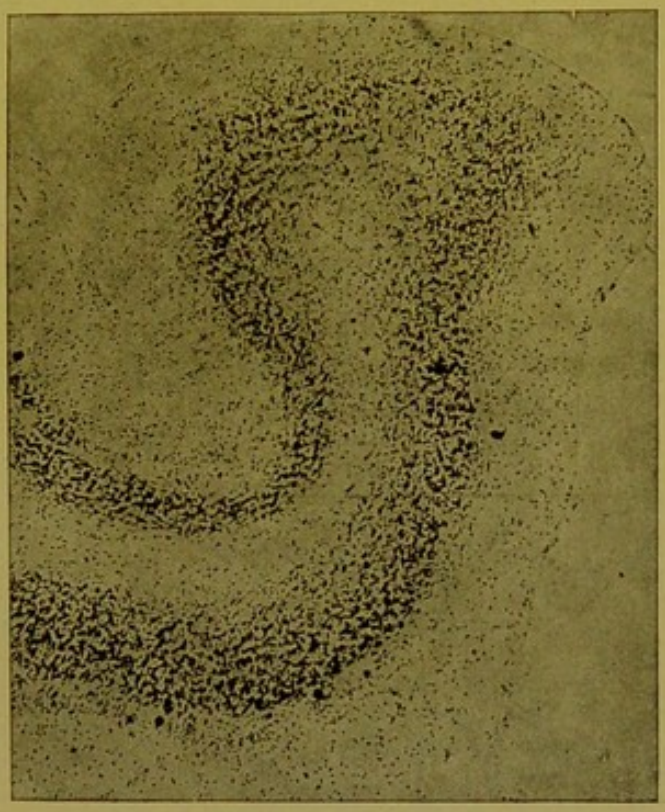


FIG. 34. Cerebellar Cortex in Paresis.

terior and lateral columns; changes in one or the other alone are less common. Fürstner found the mixed condition 73 times in 118 cases, involvement of the lateral columns alone 17 times, of the posterior columns alone 28 times; he is inclined to the opinion

[15] Westphal, Allgem. Zeitschr. f. Psychiatrie, XX, XXI; Virchows Archiv, XXXIX; Arch. f. Psychiatrie, I, XII; Fürstner, ebenda, XXIV, i; XXXIII, 939; Kinichinaka, ebenda, XL, 900; Mayer, ebenda, XLIII, 1.

that the spinal cord is affected without exception in paresis. The alterations were most severe in the lumbar and dorsal cord and gradually diminished as they were followed upward. In a few cases a diffuse or focal increase of neuroglia tissue was found. Alzheimer and other observers have demonstrated infiltration of

Fig. 35. Cerebellar Cortex in Health.

the vessels with lymphocytes and plasma cells, both in the pia and in the cord itself, especially in the fiber tracts.

Figs. 36, 37 and 38 illustrate the most frequent forms of spinal cord affection in paresis, first the usual association of lateral and posterior column degeneration, then a posterior column involvement similar to tabes and a pure lateral column affection. Besides these findings which, in their distribution, point to an

independent disorder in the cord itself, there are often tract-degenerations which are to be regarded as secondary to focal lesions in the cortex. An example is given in Fig. 39. Here,

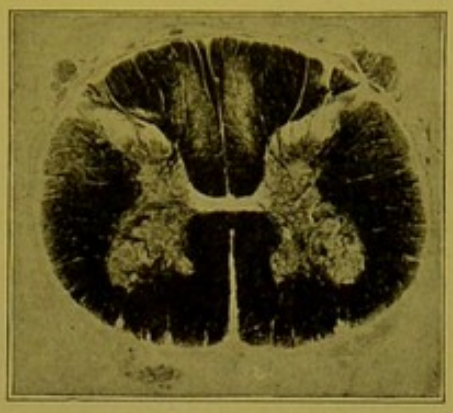


FIG. 36. Spinal Cord in Paresis. Degeneration of Dorsal and Lateral Columns.

in addition to a posterior column affection such as is found in early tabes, there is a degeneration of the lateral column on one side. In life there had been for several months a spastic hemiparesis following a paretic attack. Sometimes the anterior as well

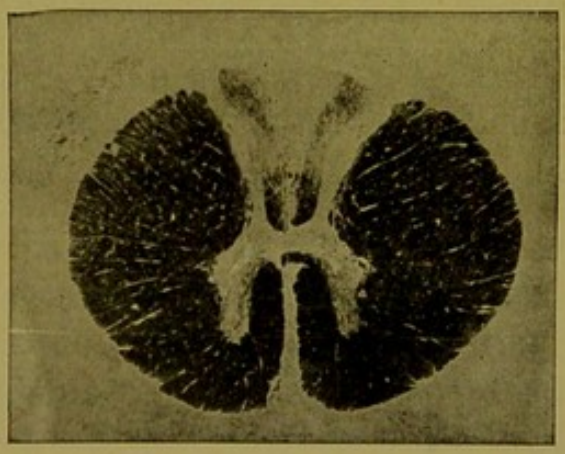


FIG. 37. Spinal Cord in Paresis. Degeneration of Dorsal Columns.

as the lateral pyramidal bundles show secondary degeneration. In a few cases the changes of syringomyelia have been found.

Hoche[16] found diffuse degenerative changes in the ventral and dorsal roots especially in the lumbar region, apparently independent of the disease in the spinal cord.

In the sympathetic system alterations such as atrophy, and pigmentary degeneration of the cells and fiber loss have been described by various writers. In the peripheral nerves[17] also, in the saphenus major, the peroneus and the long thoracic, degeneration has been found which corresponds well with our knowledge of tabes and with the frequent paralyses seen during life. In view of the scarcity of such findings Fürstner emphasizes that we are not yet justified in regarding them as belonging to paresis, but rather as dependent upon a number of other causes, alcoholism, tuberculosis, marasmus, contusions, which are capable,

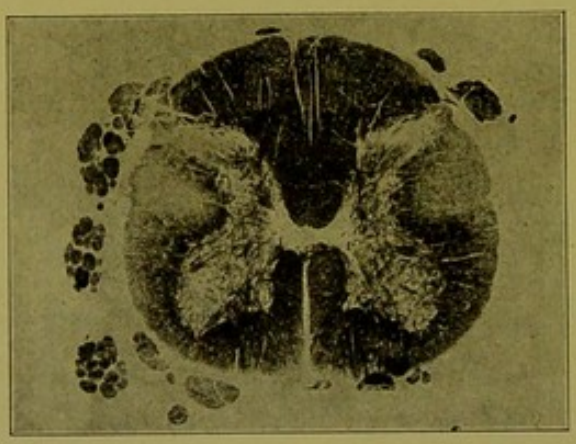


Fig. 38. Spinal Cord in Paresis. Degeneration of Lateral Columns.

as a matter of experience, of producing neuritic disease. Sciuti has found the fibrils in the peripheral nerves everywhere more or less affected. This is especially the case in the motor nerves and those in the legs show greater involvement. Stransky also has stated that signs of degeneration are more common in the peripheral nerves than in other mental diseases which are accompanied by somatic illness.

[16] Hoche, Beiträge zur Kenntnis des anatomischen Verhaltens der menschlichen Rückenmarkswurzeln, 1891.

[17] Stransky, Obersteiners Arbeiten, Festschrift, 1907.

As to the remaining organs, those alterations naturally are to be set down, in the first place, which are dependent upon the usual causes of death in paretics, namely pneumonia, tuberculosis, septic disease, pyelo-nephritis and the like. There are still a number of findings to be mentioned which, on the one hand, cannot be regarded as dependent affections and yet, on the other hand, are

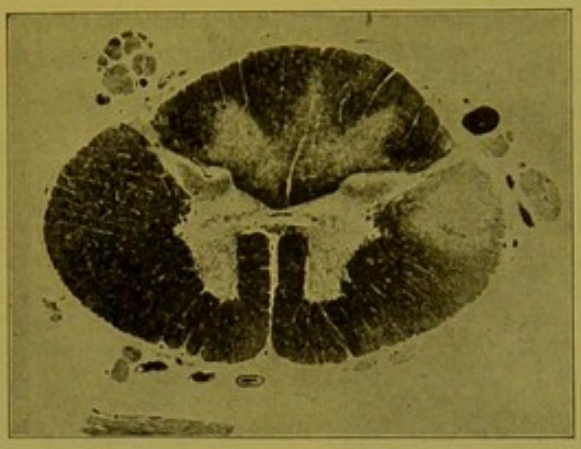


FIG. 39. Spinal Cord in Paresis. Degeneration of Dorsal and Secondary Degeneration of Lateral Columns.

so frequent that it is unlikely they are simply accidental. To these belong in the first place the widespread vessel alterations, especially the disease of the aorta, which is often met with in very severe form even in quite young persons. Straub saw it in 82 per cent. of his paretics and also in the same form in seven luetic patients. He is of the opinion that the condition is a syphilitic one which is distinguishable from the atheromatous changes of arteriosclerosis, especially by the failure of all regressive signs in the diseased vessel-wall. This assumption, defended also by Heller, although as yet difficult to prove, has received strong confirmation in the recent studies on the syphilitic origin of aortic aneurism. An idea of the changes in the aorta in paresis may be gained from Fig. 40, opposite which is a section of normal aorta for comparison, Fig. 41. We note the very marked infiltration of the media consisting of lymphocytes and plasma cells. The

spindle-shaped spots are probably degenerated vessels. In the thickened intima is a streaked proliferation of the intimal cells, especially at the surface, with disturbance of the layering.

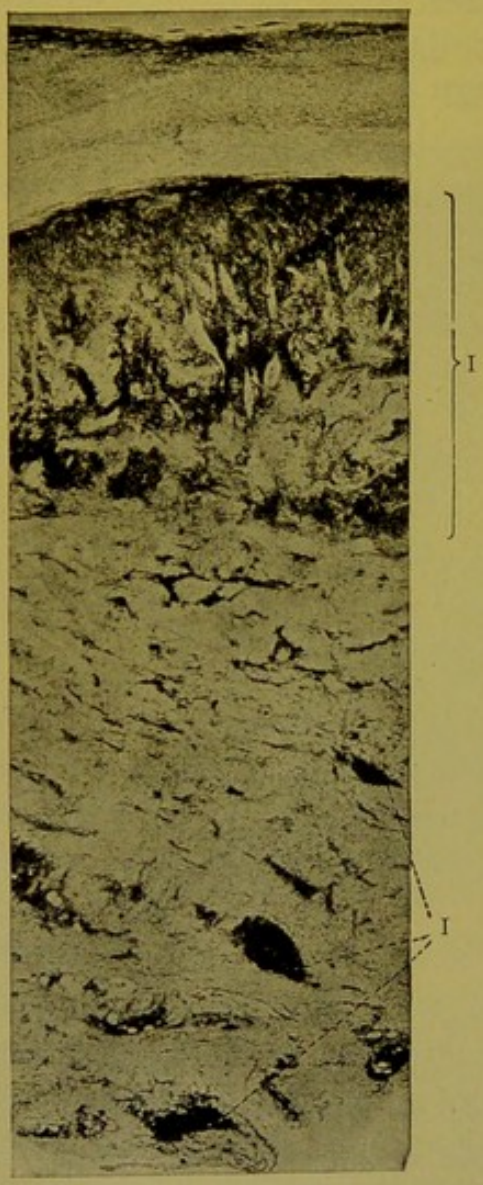


FIG. 40. Aortitis in Paresis. I. = Infiltration.

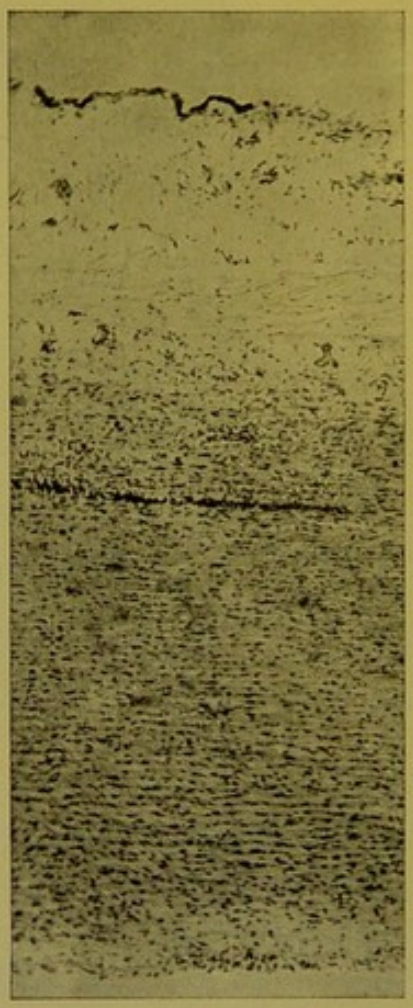


FIG. 41. Healthy Aorta.

Angiolella found periarteritic changes in the liver and kidneys in a number of patients; many other observers have likewise

remarked the frequency of kidney disease in paretics. Cardiac disease is further to be spoken of. Among 56 autopsies upon cases of paresis in Heidelberg degeneration of the heart muscle occurred 11 times, brown atrophy 4 times, fatty change 3 times, endocarditis 4 times, pericarditis once. Granular atrophy of the kidney was present in six cases; a few showed parenchymatous disease of the liver. Pilcz records similar experience. In 896 post-mortems there were 280 with aortic atheroma, 58 with brown atrophy, 227 with simple, 104 with fatty degeneration of the heart, 53 with valvular disease. Simple atrophy of the liver was found 235 times, brown atrophy 33 times, fatty degeneration 60 times, cirrhosis 8 times; arteriosclerotic nephritis was observed 19 times, simple atrophy of the kidneys 225 times, fatty degeneration 46 times; the spleen was atrophic in 227. Lukacz found aortitis in 66 per cent. of his cases, cardiac disease almost without exception and very frequent atrophic changes in liver, spleen and kidneys. Mott observed fatty degeneration of the muscles in 70 per cent., but only in cases with convulsions. Alzheimer, finally, saw in his 170 cases, fatty change in the heart muscle 71 times, in the liver 115 times; 34 showed liver-atrophy and 52 had kidney alterations. Aortitis was present in all but 34. Marcus found this condition almost without exception. It must be admitted that the relation of these numerous and irregular changes, as well as that of others which frequently occur, to the paretic process is at present very obscure, especially when we must reckon with all sorts of incidental influences (alcoholism, senility, infection, marasmus). Nevertheless it seems important that the various body viscera are found diseased to a very remarkable extent in paresis.

Anatomical Groups

The widespread and striking anatomical alterations in paresis on the one hand and the unsatisfactoriness of the clinical groupings now in use, on the other, have repeatedly brought up the

consideration of dividing the different disease-pictures on a basis of pathological findings.[18] Tabetic paresis first presents itself as a special form. In this there is clinically and anatomically an association of the tabetic and the paretic process. There is, as already pointed out, a small group of cases in which the signs of tabes commence a long time before, much more seldom, as in Tuczek's case, after the development of the paretic syndrome. In these cases the findings in the spinal cord apparently agree, as a rule, with those of ordinary tabes; we must come to the opinion, then, that we are dealing, in such cases, simply with a coëxistence of two diseases quite different and independent of one another.

This fact, however, taken with the knowledge we have now of the courses of both diseases, leads to the question whether even the spinal cord changes in paresis are not to be regarded simply as tabes, and whether dementia paralytica, as well as the development of tabes-paresis is not an affection of the brain by the same process which attacks the cord in tabes. The similarity in the cytological and serological findings seems also to speak for this view. The discussion of this theory (which led Moebius to see in paresis a tabes of the cerebral cortex) has been conducted with great vehemence.[19] The arguments in favor of it have been noticeably more on the part of neurologists (Raymond, Schaffer, Nageotte and others), those against it by psychiatrists.

I may also mention that a simple mixture of the two processes is not consistent with our present views. It is to be remembered that clinically we recognize mental disturbances in tabes which differ materially from the paretic symptom-complex and also show a different anatomical picture, further, that tabes regularly has an extremely insidious and protracted course with frequent arrests, in some cases recoveries, and only in a relatively slight

[18] Alzheimer, Centralbl. f. Psychiatrie, 1902, 52; Fürstner, Monatsschr. f. Psychiatrie, XII, 409; Gaupp, Arch. f. Psychiatrie, XXXVII, 2.

[19] Schaffer, Über Tabes und Paralyse, 1901; Cotton, Amer. Journal of Insanity, LXI, 4.

measure threatens life. All this changes so soon as the symptoms of paresis are added to those of tabes. Furthermore, the nervous symptoms of the usual dorsal cord involvement of paresis do not, in any way, coincide with those of tabes. They differ in the absence, in tabetic paresis, of lancinating pains, crises, ataxia and the peculiar sensibility disturbances. The bowel and bladder symptoms are also less marked and the eye-muscle paralyses as well as the optic atrophy, which are observed in 25 per cent. of cases of true tabes, are rare in paresis without tabes. Torkel found the last-mentioned in 2.74 per cent. of all paretics practically exclusively in the tabetic form. Crises were observed in 73 per cent. of paresis with tabes but only in 17 per cent. of the ordinary dorsal-cord paresis. This difference is remarkable in the face of the great similarity of the spinal cord changes as to nature, location and distribution.

However, there is not, as a rule, a complete agreement between pure dorsal-cord paresis and tabes, as Fürstner has held. Besides the slighter involvement of the cervical region and more marked changes in the dorsal and lumbar cord, a lesser amount of change has been shown in the posterior root zones and their surroundings in paresis, as well as an early degeneration of the endogenous fiber-bundles (comma bundle, dorso-median bundle, ventral field of the dorsal columns). On the other hand, the slight differences in the histological findings (slighter infiltration of the pia in tabes, scarcity of plasma cells in the vessel-sheaths of the spinal cord) are, in Alzheimer's opinion, attributable to the circumstance that, in general, we are dealing, in tabes, with a much more gradual process. Finally, it is still to be mentioned that the frequency of the complement-fixation in the blood and in the spinal fluid seems to be less in tabes than in paresis. On all these grounds, I have reached the conclusion that the tabetic and the paretic disease-processes show many similarities and are often associated with one another, just as we may also have a gummatous affection

along with paresis, but that the two must, nevertheless, be regarded as quite distinct. Whether, as Alzheimer thinks, it is essentially only a difference in the location of the affection, I must, for the present, express a doubt, in view of the tabetic psychoses and the difference in course and prognosis.

The clinical forms of tabetic paresis do not seem to be peculiar. I found, among the cases in which tabetic symptoms preceded the paresis by 1–8 years, that the various clinical pictures occurred in about the usual frequency, perhaps with slightly less preponderance of the expansive type. Women appear to be relatively somewhat less often affected; alcoholism plays no rôle.

Lissauer has distinguished from the usual paretic picture those atypical forms in which the disease preferably involves the posterior half of the brain and leaves the frontal portion fairly free. As a result of this distribution, all sorts of focal manifestations (asymbolia, word-deafness, mind-blindness, auditory hallucinations) play a prominent rôle, usually following paretic attacks; unilateral disturbances are frequent. The whole course of the disease is more spasmodic and, in this way, resembles that of cortical epilepsy; the deterioration is more gradual and less general. Fig. 42 is from such an atypical case of paresis. Here we find in the parietal region such a profound alteration as is encountered usually in the frontal lobes in the most severe cases. In the much thickened pia the greatly dilated veins are noticed. The most superficial cortical layer is somewhat broad and shows numerous glia nuclei. Only in the second layer are some nerve cells found, while in the third all have disappeared. In the deeper layers a few shapeless nerve cells are scattered among the numerous glia nuclei; even in the white matter there is much neuroglia increase. The vessel infiltration, on the other hand, is insignificant.

Alzheimer estimates the frequency of Lissauer's paralysis at 15 per cent. of all cases, while the typical form comprises about 80 per cent. The remaining 5 per cent. is distributed in his classi-

fication among several small groups. He distinguishes the foudroyant paresis with a picture of an acute delirium and with severe, extensive, acute alterations in the whole brain, which may be grafted onto another form which has hitherto run a slow course; then a form with especially marked involvement of the cerebellum in which there are also clinically the symptoms of cerebellar disease, and finally, rare cases in which the optic

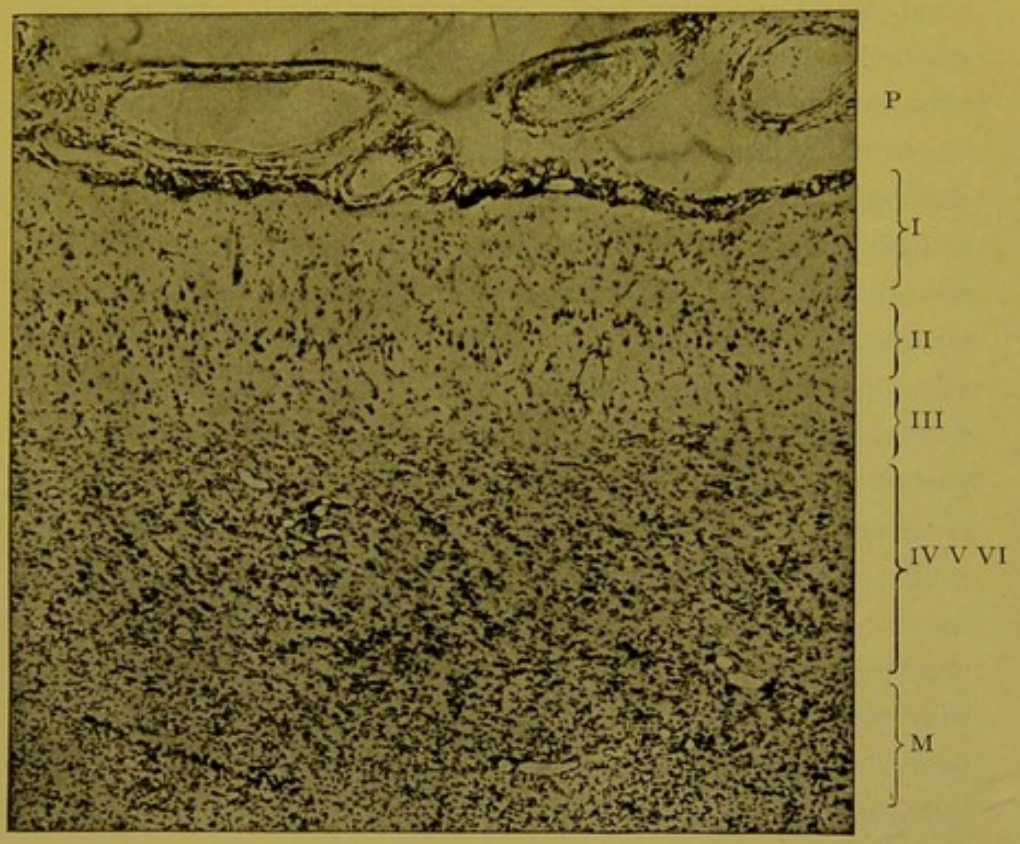


FIG. 42. Atypical Paresis.

thalamus seems to be alone or especially involved. Alzheimer is also inclined to give a special place to the senile paresis developing during more advanced years, which is characterized by excessive glia increase on account of the great loss of nervous tissue, but is marked clinically by the slight physical disturbance in comparison with the mental deterioration, approaching more the picture of senile dementia.

The so-called stationary paresis is still to be mentioned. Clinically it is characterized by its insidious development, long preservation of a certain mental capacity and relatively slight somatic signs. The patients continue to care for themselves, to read the papers, play cards, take part in events passing about them, but they have no serious purpose, live thoughtlessly from day to day, have no insight into their disease or its seriousness. Severe paralyses do not occur, speech and writing seem to be only slightly affected. One of my patients (whom I at first regarded as a case of syphilitic pseudo-paresis but whose brain after death from an intercurrent disease revealed the changes of stationary paresis), although he had had his first convulsion 20 years before and mental disorder had been prominent for 10 years, showed only a slight uncertainty and a few unimportant omissions in his writing. From its clinical course this case suggests the occasionally observed development of paresis on a basis of a previous cerebral lues. The course of stationary paresis, in spite of prolonged periods of arrest, is in general progressive; from time to time acute exacerbations of symptoms occur. Anatomically, single or scattered areas of fresh change are to be found in the cortex; one finds, however, only a slight infiltration of the vessel sheaths, a scarcity of plasma cells, absence of marked glia proliferation and of distortion of the cortical architecture. Alzheimer is disposed to give to this form also a position of some independence on account of the very slight involvement of the vessels.

Binswanger has likewise given a pathologic-anatomical grouping of paretic forms. He distinguishes a "meningitic-hydrocephalic" form with marked leptomeningitis, fiber-degeneration and widening of the ventricles, a "hemorrhagic" form which comprises the majority of the cases with subacute course and shows post mortem, besides the usual paretic changes, numerous gross and petechial hemorrhages as well as marked hyaline degeneration of the vessels. In this connection it must be remarked that,

in the experience of other writers, hemorrhages are among the rare exceptions in paresis. As a third form, Binswanger mentions tabes-paresis with prolonged course and especial degeneration of the fibers running to the posterior regions of the brain. Finally also, a "peripheral-neuritic, visceral" form with severe general and local disturbance of nutrition, visceral pains and paresthesias which has not as yet been distinguished on anatomical grounds.

Fischer has evolved still another classification partly from an anatomical, partly from a clinical standpoint. Besides the typical form, he distinguishes Lissauer's focal paresis with its various localizations in the cortex of the posterior portion, in the cerebellum or in the thalamus, then the "atypical paresis" which comprises the catatonic, senile, foudroyant and some of the tabetic forms, and finally, epileptic and stationary paresis. The further clinical and anatomical study of this subject must show how far relations between brain changes and clinical symptoms can be established during life, so that we can predict the autopsy findings and by them, in turn, explain the clinical symptoms.

CHAPTER IV

Etiology

Since dementia paralytica has become well known only within a relatively short time, the peculiarity and severity of the clinical picture warrant the opinion that the disease has only succeeded to its present frequency during our age. Möbius has referred to the striking fact that no contemporary of the eighteenth century has described paretic symptoms in any prominent man, although, as a matter of fact, many of them suffered from mental disease. In contrasting this with later experiences of the nineteenth century where Junot, Lenau, Schumann, Donizetti, Rethel, Makart, Nietzsche and Maupassant undoubtedly died of paresis, it would seem that the disease must at least have been much less frequent in the past. At the present time the average admission of paretics to our hospitals for the insane is about 10–20 per cent., yet this rate is subject to extreme variation.[20] During the last decade the percentage of paretics has increased considerably in a number of institutions, as has been demonstrated by Krafft-Ebing; more rarely has the contrary been noted. At all events it is extremely difficult to state what rôle the more or less extended delimitation of paresis plays here. Personally I do not consider the diagnosis of paresis at all so certain, especially when it is not founded on observation of its entire course or upon cytological and serological findings, that I can draw conclusions from the statistical statements of various observers concerning the frequency of the disease at different times. Such a comparison would lead to the false conclusion that the disease has become considerably less

[20] Wollenberg, Arch. f. Psychiatrie, XXVI, 472; Gudden, ebenda, 430; Junius und Arndt, ebenda, XLIV, 249; v. Krafft-Ebing, Jahrb. f. Psychiatrie, XIII, 127; Hirschl, ebenda, XIV, 321; Bär, Die Paralyse in Stephansfeld. Diss. Strassburg, 1900.

frequent of late, because I have become more and more careful in the diagnosis of uncertain cases after a series of false diagnoses.

Nevertheless a certain increase of paresis may be admitted as highly probable. The experience, especially of larger cities, proves this. There the figures are so large and so continually growing, as for instance the rate of Berlin and Munich, where the male paretics amount to 36 per cent., and in the Charité at Berlin where they reach 45 per cent., that the errors just mentioned are of little consequence. There are twice as many paretics in the city asylums as in those of the country districts. This fact, considering the rapid growth of the city population, makes an increase of paretic affections more probable. The percentage of the admitted paretics in Berlin is at least four times as great as in Westphalia. In Heidelberg, with its industrial environs, paresis is much more frequent than in Freiburg, where the admissions are mostly country people. The number of paretics admitted from Tübingen is given by Jolly as 5.2 per cent. and for Kiel 11.2 per cent. The lower Alsace without Strassburg furnished 10.6 per cent. and Strassburg alone 40.3 per cent. of paretics. In estimating the figures of Altscherbitz, Hoppe finds one case of paresis occurs to 3,000 city dwellers but among country people only one in 9,000. The number of paretics in the essentially rural districts of Tyrol is estimated by Eisath as only 5.4 per cent. of admissions to institutions. Paresis is considerably more rare in Sicily than in North Italy. It has been stated by Steward that an increase of paresis has been observed in the mining and industrial districts of England, while a decrease has appeared in the agricultural communities. Again one finds a relatively high number in the private institutions which are filled from the city population. Kiss found in a Hungarian private hospital 41.5 per cent. of paretics among the male patients.

The number of paretics in proportion to the total admissions often decreases; thus, in England, it sank between 1888 and 1897 from 8.7 per cent. to 7.8 per cent., similar statements have been

made by Junius and Arndt from Dalldorf. However, these experiences may very probably be based upon the increase of other forms of insanity (for instance alcoholism) or perhaps entirely upon their facilitated admission.

Of much greater significance for the understanding of paresis are the extraordinary differences in its frequency in different countries.[21] While in France, England, Italy, Austria, the Netherlands, Switzerland, in Western Russia and the Eastern United States almost the same relations obtain as in Germany, the percentage of paretics in Spain, according to Rodriguez-Morini, is only 5–6 for men and 0.9 for women. In Scotland, Woods states 4 per cent. of admissions are paretics, in Ireland 1 per cent. In Canada the number is estimated at 1.65 per cent., in Chile 2.8 per cent. In Norway, according to Vogt's personal observation, paresis is so rare that sometimes there is not a single case in the institution of 330 beds at Gaustad. In Iceland paresis is entirely unknown among the natives.

The condition in Bosnia and Herzegovina as described by Glück, Hödlmoser and Kötschet has aroused especial interest. The latter states that in Sarajewo, in spite of the widespread prevalence of lues, only 0.65 per cent. of the natives admitted to institutions are paretics, while the percentage for foreigners is 9. In the neighboring country, Croatia, the admissions for paresis reach 16.5 per cent., but among the patients from Bosnia and Herzegovina only 2.4 per cent. Hödlmoser finds arteriosclerosis and aneurism rare in the latter countries, while hysteria and neurasthenia are very common. In Algiers paresis is very seldom seen in natives; Rüdin, even with careful search in all the hospitals and asylums, could find only two undoubted cases. Among the Arabs in Cairo, however, Marie found 6 per cent. of paretics. Mongeri reported from Constantinople that Turks are little inclined to develop paresis; the same applies to Spanish Jews.

[21] Scheube, Die venerischen Krankheiten in den warmen Ländern, 1902; Hödlmoser, Wiener klin. Rundschau, 1904, 13; Kotschet, Wiener med. Wochenschr., 1904, 24.

The extended inquiries of Scheube have shown further, that paresis is apparently unknown in British East Africa, Uganda, Zanzibar, Kamerun, Togo, Samoa, the Marschall Islands and in Nicaragua. It is uncommon in Asia Minor, British India, Siam, China, Corea, Abyssinia, Natal, on the Gold Coast, in Madeira, the Fiji Islands and Haiti. It seems to be more frequently met with in Cuba, Jamaica, British Guiana and California. I was, myself, unable to find a single case of paresis among the natives in the insane hospital of Buitenzorg in Java, but a brain from an undoubted paretic Javanese was sent to us from there later. Moreira saw 2.76 per cent. of paretics among patients from Rio. In Japan paresis seems now, at least, to be as prevalent as in the civilized countries in Europe.

One may be inclined at first to attribute these remarkable differences to racial peculiarities. A circumstance that speaks in favor of this is that in Java and Algiers the Europeans are just as subject to paresis as at home, that in Constantinople it is far more frequent in Armenians and especially in Greeks, than in Turks, that in Bosnia the natives and foreigners show a different susceptibility to paresis, that in Cuba the negroes are less affected than the whites and that the Brazilian Indians apparently do not have the disease. Further consideration shows, however, that, on the one hand, great differences are seen between peoples from the same racial stock (Iceland and Norway—Denmark, Bosnia—Croatia, Hungary—Turkey) and, on the other hand, very different races exhibit the same tendencies regarding paresis. Thus we see the disease frequently in Slavs, Romans, Germans, Hungarians, seldom among Celts, Turks, Abyssinians, Indians, Negroes, Malaysians, etc.

Just as little can the kind of food be advanced as an explanation, in view of the vegetable diet of most of the peoples who are resistant to paresis. The Turks and Spaniards eat meat like the Armenians, the same is true of Icelanders and Norwegians; the inhabitants of Bosnia employ essentially the same food as the

Croatians; the Japanese are subject to paresis in spite of prevailing vegetable diet. Nor can climate play any important rôle, since in all latitudes there are countries in which paresis is rare and, what is more significant, Europeans in other climates are just as apt to become paretic as in their home countries. We are drawn much rather to the conclusion, from the considerations mentioned, that paresis stands in some causal relation with the general habits of life, such as those which prevail in middle Europe and which have spread with Europeans to other lands.

As a matter of fact there are observations which indicate that other nationalities have become susceptible to paresis through contact with European modes of living. French authors have mentioned that Algerian natives may become paretic if they are "Europeanized." One of Rüdin's Algerian paretics was a coachman and guide for foreigners, the other a prostitute. Moreira made the observation that negroes coming from Africa to Brazil do not have paresis but that native negroes and those of mixed blood are susceptible. Among the civilized Arabs of Cairo, paresis seems to be much more frequent than among less cultivated Algerian branches of the race. Paresis was a great rarity in North American negroes a few years ago, while now they are relatively more prone to the disease than the whites. An inquiry very kindly undertaken for me by Hoch in New York, concerning the patients of seven large North American insane hospitals, showed that the average rate for paresis was 11.2 per cent. for men and 3 per cent. for women, while among the negroes it was 28 per cent. for men and 8.1 per cent. for women. The North American Indians, who are gradually dying out in their reservations, also suffer severely from paresis.

Of the two sexes, the male is represented among the paretic patients about 2–5 times as often as the female. In earlier decades the ratio was placed as 1:7 or 1:8. In the two decades between 1877 and 1897 Buchholz saw the relations change from 1:7.2 to 1:6.5, Soukhanoff saw it vary from 1:6.7 to 1:5.1,

Epstein from 1:5.2 to 1:3.9 and Greidenberg, Moravczik and Hieronymus also mention the relative increase of paresis in women, while Steward claims a decrease in England. Although coincidence may play a large rôle in these figures, still the relative number of cases in women is certainly increasing in Germany, especially in the institutions receiving from large cities. The large municipalities appear, therefore, not only to increase markedly the frequency of paresis but also to eliminate, to a certain extent, the difference ordinarily existing between the sexes. In women of the lower and in men of the higher classes, paresis is relatively more common; in women from the higher walks of society the disease is very rare. The proportion of the two sexes in an Hungarian private hospital was, according to Kiss, 30:1, in the Budapest clinic 5.6:1; in the private institution of Oberdöbling it was 13.8:1 (Obersteiner).

In this connection it is worth while also to consider the relations existing in other countries. In Spain, according to Rodriguez-Morini the proportion of the two sexes is 9–10:1, in Greece 15.2:1, in Rio 18:1. in San Paolo 30:1. Turkish women of the lower classes and Greek women of the higher classes are practically never affected. It seems therefore that the difference in the proportion of the sexes is greater where paresis is less common, but that other influences must also come into play. Of the clinical forms, it is my experience that women usually have demented paresis. Expansive cases are relatively uncommon.[22] I have not, as yet, been able to confirm what various observers have reported, that the average duration is longer in women. Bär found the duration of the disease in men to be 33, in women 47 months, Sprengler 30 and 41½, Greene 15 and 23 months respectively.

A table dealing with the different ages is given in Fig. 43. Here the age of onset in 530 men and 203 women is given in 5-year periods. We know that the greatest frequency for men

[22] Jahrmärker, Allgem. Zeitschr. f. Psychiatrie, LVIII, 1.

lies between the 30th and 50th year, or more closely, between the 35th and 45th. Before the 30th year paresis is rather uncommon, while after the 50th year it is still fairly frequent. Even in the seventh decade a few cases are seen. In women these conditions are, in general, similar. In them also the period of greatest frequency is between the 30th and 50th years, but more cases seem

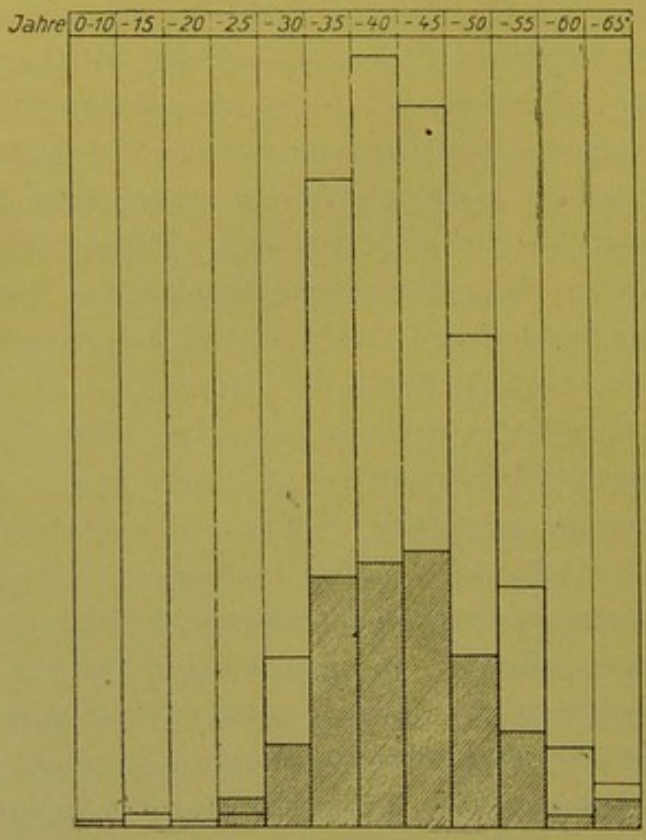


FIG. 43. Representation of the two sexes in paresis at different ages. Shaded portions, men.

to fall between the 40th and 45th year than in the previous decade. Between the 20th and 25th year it is not only relatively but actually more frequent than in men, and even in the next five years there is much less preponderance of the male sex than in the following periods. A similar condition is found between 50 and 55 years and between 60 and 65 years. Women, therefore, become affected somewhat later on the average than men. The

climacterium may have a certain influence. One, however, also finds a group which develop very early and finally the proportion of women is somewhat higher also in the later periods of life. Although the number of cases is rather small, the table agrees so completely with the findings in Dalldorf (Junius and Arndt, 1,304 cases) that there is no doubt of its utility.

Paresis of the juvenile years occupies a certain position of independence.[23] Clouston described the first case in 1877, since which time a large number of similar cases have been reported. To-day juvenile paresis can hardly be called more than uncommon; I can often demonstrate 4 cases at once in the clinic. Whether such cases are actually more frequent than formerly remains to be proved; its clinical picture is often so atypical that it was doubtless frequently overlooked in earlier times. The disease begins usually in the years of development, but often much earlier, even at 5 or 6 years. The children are often inferior from infancy, both physically and mentally; in other cases, however, they develop normally for many years and gradually show signs of the disease just as adults. It is occasionally apparent that the paresis is grafted onto a previous syphilitic brain disease. The children are inattentive, weak in memory, awkward, do not progress in school and become gradually demented.

The symptom-picture, as a rule, is that of simple demented paresis; only exceptionally do childish ideas of grandeur occur. Hypochondriacal ideas are also uncommon, excited states somewhat more frequent, in which the patients show senseless crying, do not remain in bed, throw themselves about, grasp at imaginary objects. They gradually become uncertain in gait, then

[23] Alzheimer, Allgem. Zeitschr. f. Psychiatrie, LII, 3; Thiry, De la paralysie progressive dans le jeune âge, 2898; Hirschl, Wiener klin. Wochenschr., 1901, 21; v. Rad, Arch. f. Psychiatrie, XXX, 82; Mingazzini, Monatsschr. f. Psychiatrie, III, 53; Frölich, Über allgemeine progressive Paralyse der Irren vor Abschluss der korperlichen Entwicklung. Diss., 1901; Watson, Arch. of Neurology, London county asylums, II, 621.

bed-ridden and unclean. Contractures develop first in the legs and then in the arms. Often one observes continuous sucking or chewing movements. Convulsions seem to be very frequent; they are mostly of an epileptic character and may be repeated day after day, without any apparent after-effect. Fig. 44 gives, by weeks, the number of convulsions occurring in a case of juvenile paresis. Besides the extraordinary number of attacks,

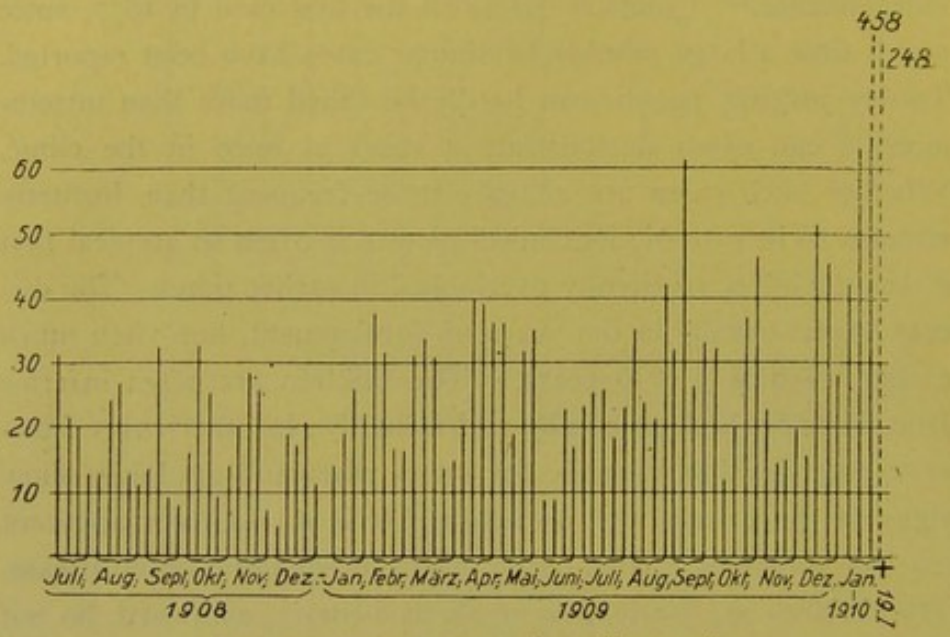


FIG. 44. Convulsions in juvenile paresis.

one notices also the fairly regular fluctuations and a gradual increase in frequency. In the last week their number rose to 458 and on the last day there were 248. Many juvenile paretics pass as cases of "idiocy with epilepsy" until the cytological and serological examination with the characteristic result, or the further course or microscopical examination of the brain make the picture clear. One not infrequently encounters such cases in institutions for idiots. The Babinski reflex is often demonstrable for a long time; sometimes a permanent dorsal flexion of the great toes results, as in Fig. 45. Optic nerve atrophy appears to be frequent.

The course of the disease is generally prolonged and episodic; the duration is usually 3–4 years or more; in one of our cases, the one from which the diagram of convulsions was taken, it was over 9 years. The findings post mortem are the same as in

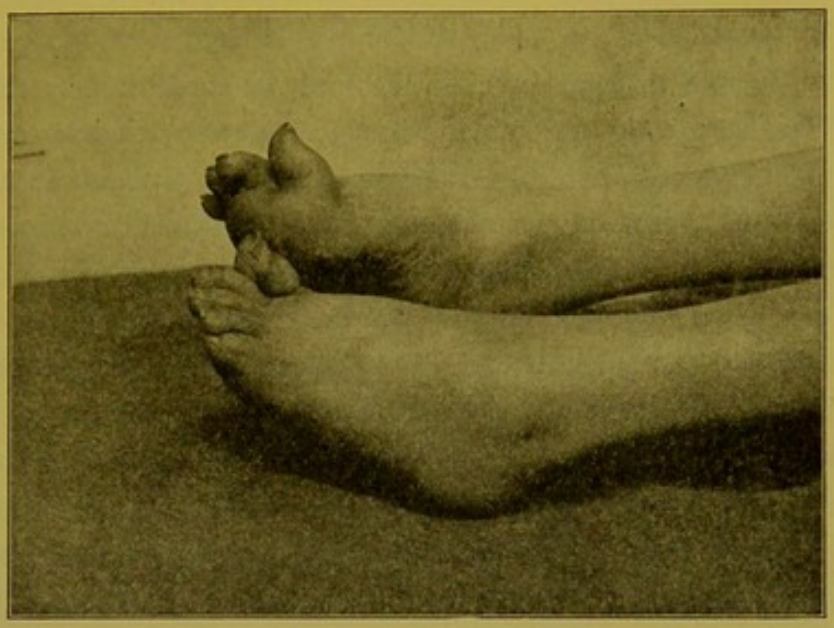


Fig. 45. Permanent Dorsal Flexion of Great Toes in Juvenile Paresis.

paresis in adults. Straüssler has found that a striking number of multi-nucleated Purkinje cells occur in the cerebellum, an indication of an early-beginning developmental disorder. Fig. 46

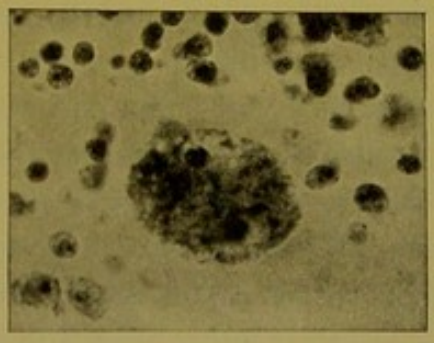


Fig. 46. Double-nucleated Purkinje Cell in Juvenile Paresis.

shows a double-nucleated Purkinje cell from the cerebellum of a juvenile paretic. Trappet also observed syncytia of the nerve-

cells. Joffroy found the carpal and metacarpal bones not yet ossified in a congenitally syphilitic paretic of 23 years.

What gives especial significance to juvenile paresis is its clear etiological connection. In the first place it shows us that of all the causes which may be assigned in adults, not a single one can be regarded as an essential for the development of paresis. In the juvenile variety, however, lues may be shown with remarkable clearness to be the common basis. Indeed Alzheimer found that a dependence upon lues was certain or very probable in about 70 per cent. of the cases. Among 20 cases Hirschl obtained 17 certain and one probable history of inherited syphilis, Mott found evidence of lues by examination or in the anamnesis in 80 per cent. of 22 cases. Occasionally an initial infection in early childhood can be proved. Often the parents suffer from tabes or paresis, the brothers or sisters from obvious congenital syphilis. In the cases observed by us there was, without exception, a pleocytosis in the spinal fluid and Wassermann reaction in this and in the blood serum.

It is an important fact that congenitally syphilitic children, especially those of paretic parents, sometimes show complement-fixation in blood and spinal fluid without, themselves, having any signs of paresis. We may conclude that, in general, the development of paresis is long preceded by the appearance of the Wassermann reaction, although Eichelberg and Pförtner have described a case of paresis with Wassermann reaction in which this test had been negative a year and a half before. In any case the conjecture is permissible that congenitally syphilitic children with the serological indications of paresis are in danger of developing that disease after the lapse of the usual space of time. The future must show whether this fear is borne out in the children so far examined and whether therefore we are in a position, on the basis of the complement-fixation, to speak of a "preparetic" stage of the disease.

External evidences of hereditary syphilis are not infrequently

seen in juvenile paretics. One patient observed by us had a syphilitic alopecia; two showed well-marked Hutchinson teeth. It is remarkable that the two sexes are equally represented in juvenile paresis; the factors which determine the different susceptibility of the sexes in adult life must therefore be sought in the influences arising in later years.

Unmarried persons seem to be more prone to paresis than married. It is not very exceptional that a man and wife are affected by paresis or tabes at the same time or shortly after one another. Räcke[24] collected 69 such observations. In 27 cases both had paresis, in 22 cases tabes; in 14 the man had paresis, the woman tabes and in 6, the reverse. Junius and Arndt described 31 cases of conjugal paresis, as well as 6 couples with paresis in the man and tabes in the woman and one with the reverse. They mention also a paretic who had a paretic daughter, while both his first wife, divorced, and his second, had tabes. It seems from this that the male sex tends more to paresis. The interval between infection and the development of paresis is, in Pilcz's opinion, shorter in men, the duration of the disease longer. Plaut has made the valuable observation that five wives of paretics and two husbands of paretic women gave complement-fixation in the blood without showing any symptoms of syphilis.

Among the different vocations, officers, merchants, firemen, railroad conductors occur relatively frequently, while Quakers and Catholic priests are very seldom affected. Bennecke states that non-commissioned officers of the army have the disease frequently, and very early in life, especially commissariat officers. Caboureau found among his patients of priestly calling only 1.9 per cent. paretics against the general relations of 17.5 per cent. Krafft-Ebing also did not see a single case of a Catholic priest among 200 paretics, while among insane army-officers 90 per cent. had paresis. Kundt observed 17 cases of mental disease in

[24] Räcke, Monatsschr. f. Psychiatrie, VI, 266; Mönkemöller, ebenda, VIII, 421; Junius and Arndt, ebenda, XXIV, 10; Mayer, Arch. f. Psychiatrie, XLV, 964; Pilcz, Neurol. Centralbl., 1909, 50.

priests without a single instance of paresis, while among 13 soldiers there were 8. Bouchard found among his men patients 28 per cent. of paretics, among 288 catholic priests only 2.4 per cent. Paresis has apparently not yet been observed in nuns. The juvenile paretics of the female sex are strikingly often prostitutes. Hollós found paresis five times as common in prostitutes as in other women, ten times between the 26th and 36th year. Hübner saw, among 43 insane women who had been prostitutes, nine cases of paresis, three of tabes and two of lues cerebrospinalis, while the percentage of paresis for the whole women's division was 13.5. Among the deaths of prostitutes 58.5 per cent. are due to paresis, among the deaths of other women patients only 16.7 per cent. Junius and Arndt saw among the women patients in Dalldorf, 11 per cent. of prostitutes, while this class only formed 1.7 per cent. of the female population of Berlin at the same time.

A further striking fact is that among paretic families childlessness, abortions and premature births, as well as deaths in early infancy, are extremely common. Hübner found 45.7 per cent. fruitless marriages among his paretics and only 14.4 per cent. among the other psychoses; in 5.7 per cent. (2.5 per cent.) the only pregnancies terminated in abortion. Régis held that three fourths of the marriages of paretics were without issue. Ricard investigated 49 paretics. Pregnancy occurred 152 times with 61 abortions; 22 of the children which were born alive died before, 2 after the 3rd year; 67 remained alive. Hirschl, who found childless marriages in only 17.7 per cent., states that 175 male paretics had 233 living children; besides these there were 106 abortions, 30 stillbirths and 154 children which died early, mostly in convulsions.

A glance over our Munich statistics gives the following table:

Par tic Marriages	Number	Childless	Abortions or Still births	Children Dead	Children Living
Husband paretic	184	33	122	185	304
Wife paretic	73	13	101	96	99
	257	46	223	281	403

The number of unfruitful marriages of paretics, therefore, in our experience 16 per cent., agrees with Hirschl's and differs from Hübner's and Régis' statements, while the figures for the whole of Germany are, according to Mendel, 10–15 per cent. Our figures show, however, that the offspring of these marriages are fewer in number than the parents—an attempt, then, at extinction. If so small a number of instances allows of drawing a further conclusion, it seems that paresis in the mother is more fatal to the bearing of children than that in the father.

The surviving children of paretics[25] show, from the studies of Ricard, Semper and Scholten, in very large numbers, the signs of mental and physical inferiority, such as infantile palsies, epilepsy, hysteria, various grades of imbecility, lymphatic diathesis, glandular swellings, adenoid hypertrophy, diseases of nose, ears and eyes, muscular weakness, rickets, anomalies of development. Many become paretics. Of 124 children of paretics who could be examined by us, 66 were physically well and strong, 52 weakly and in a poor state of nutrition (21 rachitic), 6 afflicted with gross bodily deformities. Eight had prominent mental disorders (4 paretics), while 19 were mentally sound, 31 excitable and nervous, 4 quiet and retiring. Scholten found 36 out of 111 children of paretics suffering from nervous disturbances. It can be shown that the frequency of such diseases in the children born during the last 10 years before the outbreak of paresis reaches 48.9 per cent., for those born earlier, only 13.6 per cent.

It seems much more important to investigate closely the nature of the diseases which are prone to occur in families of paretics. The first significant fact is that sometimes parents and children are paretics. Amélién has met with this six times in 238 cases; once two brothers had paresis and a sister tabes. Rieger observed a family, which I also had known, in which

[25] Ricard, contribution à l'étude de la déscendence des paralytiques généraux, Thèse, 1900; Semper, les enfants des paralytiques généraux, Thèse, 1904.

father, son, daughter and granddaughter had paresis. In the experience of Pilcz paresis begins earlier in the descendants of paretics, but lasts longer. It is transferred by preference to the same sex. In juvenile paresis, the same type of disease is very common. Frölich saw paresis in the parents in 16.4 per cent.; in an additional 13 per cent. there was either tabes, hemiplegia or apoplexy in the parents. The transference seems to be preferably to the same sex. Ricard calls attention to the difference between the progeny of paretics and those of alcoholics, the latter usually having very numerous children with all sorts of mental and nervous diseases.

The influence of heredity is, as most observers agree, less prominent in paresis than in other mental diseases, but seems to play a somewhat greater rôle in juvenile cases. My own investigations, in those cases concerning which reliable histories could be obtained, gave 50 per cent. of hereditary basis, more in the male sex than the female. Schlegel found defective heredity in 56.6 per cent., Hoppe in 41.9 per cent. in men, in 39.2 per cent. in women, Junius and Arndt in 39 per cent. and 36.1 per cent. respectively. All these rough figures can hardly serve to base an opinion regarding the actual causal significance of heredity for paresis. To be more specific, the histories of 316 paretics showed tabes or paresis of father or mother nine times, twice in uncles or aunts; once the father and grandfather were paretics. In 4 instances brothers or sisters were probably or certainly paretic. Apoplexy in the parents occurred seven times, in their brothers or sisters twice; once both parents were apoplectic. Epilepsy or cerebral lesions were present twice in the parents, seven times in their brothers or sisters, mental diseases ten times in the former, twelve times in the latter, psychopathic states thirteen and three times respectively, drunkenness eight times and twice respectively. It appears to me from this admittedly too small number that there is some justification for the opinion that organic cere-

bral lesions in the antecedents and relatives of paretics play a constant rôle.

Recently, Näcke has, without, I think, sufficient justification, again strongly brought forward the influence of heredity in paresis and has used the term "born paretics"; Raymond also holds that an abnormal brain is a prerequisite of the disease. Pilcz, on the contrary, saw heredity in only 18.7 per cent. of his cases. He came further to the conclusion that degenerative stigmata are very uncommon in paretics. In particular he found that the excess of the arm-span over the body length, which is the rule in degenerates and epileptics, only occurred in 37.6 per cent. of paretics. From this the conclusion may be drawn that degeneracy affords a certain protection against paresis; the disease also has a longer course in such persons. I regard this reasoning as highly improbable. On the contrary it is, as Alzheimer believes, quite conceivable that a psychopathic constitution can, especially in the early stages, cause such an admixture of unusual symptoms that recognition of the disease is difficult.

We must regard syphilis[26] as the only essential cause of paresis. It occurs with striking frequency in the past histories of paretics, even though existing evidences of the disease are relatively seldom demonstrable. It must be admitted that the opinions on this point vary greatly,—after Sprengeler's statistics, from 1.6 to 93 per cent. My own earlier publications, agreeing with the experience of Gudden in the Charité, gave, in men, an assured history of syphilis in about 34 per cent. of the cases. In many others also there was a certain probability of infection. Later it increased to 50 per cent. and finally, if one adds thereto the probable cases, it reaches 78 per cent. Surprising results attended the procedure of asking each paretic, not if he had a chancre but

[26] Stoddart, Journ. of Mental Science, 1901, 441; Fournier et Raymond, Paralysie générale et Syphilis, 1905; Hudovernig und Guszmann, Neurol. Centralbl., 1905, 3; Plaut-Fischer, Allg. Zeitschr. f. Psychiatrie, LXVI, 340; Vorberg, Dementia paralytica und Syphilis, 1906.

when he had it, where it was located and how treated. I have not obtained such satisfactory results in women; if one pays especial attention to the occurrence of abortions one gets nearly the same relations as in men. Wollenberg asserts that a half of his women patients have certainly or probably had lues. Sprengeler found definite history of lues in 41.5 per cent. of paretics, Räcke in 57.3 per cent., with 20.9 per cent., besides, with probable lues. In the newer statistics the number has, in most instances, risen steadily. Torkel gave earlier 42 per cent., of late years 51 per cent. of luetic histories, in tabes-paresis, an increase from 64 per cent. to 75 per cent. Hougberg comes to 86.9 per cent. from a previous 75.7 per cent. Alzheimer announces that in a carefully conducted examination of patients of the better classes in Frankfurt, earlier lues was established in more than 90 per cent. Hirschl found, among 200 patients, 6 per cent. with positive indications of earlier syphilis. From the early history of 175 paretic men he found that 56 per cent. had certainly and 25 per cent. had probably had lues, while only 19 per cent. failed to give such data. Similar experiences have been published in large numbers.

It is, nevertheless, very difficult, in case the initial lesion is unnoticed or hidden, to make even a few positive statements as to earlier syphilitic disease. Uncultured people, therefore, usually show the smallest numbers; Marcus was able to obtain a history of lues in 76 per cent. of his patients of the higher classes but only in 26 per cent. of laborers. All the numbers found in paresis, therefore, mean only the lowest limits. It must be taken into account that, from studies in syphilis in monkeys, an infection may occur perhaps even without primary lesion. A comparison of the paretic and non-paretic mental disorders is especially instructive. Eisath found in the former a positive history of lues in 27 per cent., doubtful in 34 per cent., in the latter 1.3 per cent. positive, 1.8 per cent doubtful. Hoppe found among paretics 34.6 per cent. in men, 23.5 per cent. in women with his-

tory of syphilis, among non-paretics 10.9 per cent. in men and 2.6 per cent. in women.

Experiences of the sort just described first led Esmarch and Jessen in the year 1857 to the consideration of a causal relation between syphilis and paresis, a view which was confirmed in 1860 by Steenberg and in 1863 by Kjellberg. The controversy regarding it has continued until the present day, with the result that it has gradually gained a firmer footing. Since the statistics from the earlier histories of paretics always leave a certain number of cases in which lues is unproven or even, according to the observer, "excluded," a general opinion arose that, although syphilis plays an important rôle in the development of paresis, it cannot be the sole cause, as it seems to be absent in many cases. There must, rather, be other deleterious agents which prepare the way but which can, even by themselves, under certain circumstances, produce paresis. As such, alcohol, emotional strain or excitement and exhaustion have been brought forward.

These views are offset to a certain extent by the fact, as shown by Hirschl, that no less than 36.5 per cent. of patients with tertiary syphilis are unable to give any data regarding earlier luetic symptoms. Hudovernig and Guszmann also state that 42.3 per cent. of women with tertiary lues recall nothing of infection. Krafft-Ebing[27] further described the experiment of an unknown physician of inoculating syphilitic virus into nine paretics in whom no history of infection could be obtained. None of these cases developed secondary symptoms. From this it may be concluded that lues, in great probability, already existed in those patients. The rarity of primary lesions in paretics, although often exposed to infection during excited states, must be regarded in the same light. Cases of re-infection have nevertheless been reported by Kiernan, Morselli and Garbini; the interpretation is, to be sure, also allowable that an earlier existing lues was fully healed.

[27] v. Krafft-Ebing, Die Ätiologie der progressive Paralyse, Arbeiten II, 12, 1897.

The last link in the chain of proof of the syphilitic origin of paresis is closed through the cytological and serological studies. Both have shown us that in paresis we regularly find those conditions which are characteristic of syphilis,—increase of cells in the spinal fluid and occurrence of plasma cells and complement-fixation in this fluid and in the blood serum. The exceptions, after careful and repeated tests, are so few that they cannot be seriously considered; at present they are, for complement-fixation in the serum, hardly more than ½ per cent. Complement-fixation is admittedly not confined to syphilis and paresis (and tabes), but is also observed in a few other diseases (leprosy, malaria, sleeping-sickness), which, however, do not come into practical consideration here.

We can, therefore, to-day with greatest certainty declare that syphilitic infection is an essential for the later appearance of paresis. Only from this viewpoint can we explain the many peculiarities in the distribution of the disease, especially the difference between city and country, its rarity in women of the upper classes and in catholic priests, its frequency in officers, merchants, prostitutes, and the occurrence of conjugal paresis. It must, however, be mentioned that the husband and wife may have contracted their lues from different sources. Garnier published an observation of this sort. The different ratio of the sexes also points to a relationship to lues. The frequency of syphilitic infection in men and women is, according to Blaschko, in the proportion of 4:1, after Fournier, 8:1, so that women are now apparently very prone to develop paresis. The fact that women, on the average, develop the disease later than men is attributable to their becoming infected usually after marriage, men before marriage. The relatively greater frequency in very young and in older women makes it probable that lues was contracted in the former before marriage, in the latter, during married life. More striking is the difference in the figures in juvenile paresis which is not yet influenced by the difference in life-habits

of the sexes. Finally we must refer to the close clinical relationship of paresis to tabes and lues cerebrospinalis (husband and wife, brothers and sisters, tabes-paresis, endarteritic and gummatous processes in paresis), also to the characteristic degeneracy of the descendants, which agrees entirely with congenital syphilis.

A certain difficulty confronts the question of the etiological relationship of lues and paresis, in the fact that, in many lands, the first is very common but the latter extremely rare or unknown. Bermann found among 15,000 patients in Bosnia, 464 who were syphilitic, but he saw no case of tabes and only one of paresis and this was in a man from another country. Ostrowskich similarly states that in Persia among 7,000 patients he encountered much syphilis but neither paresis nor tabes. In Abyssinia, 80 per cent. of the inhabitants are luetic and still there is apparently no paresis. In Turkey, also, especially on the Black Sea Coast of Asia Minor, in Algiers and in many other lands whose natives hardly know paresis, syphilis is very common. Obviously, then, certain general conditions must be fulfilled if the syphilis is to produce paresis.

One may first think of different potentiality of the syphilitic virus. Even among us, not all syphilitics, but only a small fraction of their number, develop paresis. Indeed tertiary symptoms only appear in a small portion of the cases of syphilis, according to Marschalko in 8 per cent., while other authors' estimates vary between 6 per cent. and 22 per cent. Mathes who gathered the facts concerning 698 persons, of which 568 had had secondary, 130 tertiary symptoms, found that eight had become paretics. Hudovering and Guszmann followed the fate of 50 persons with late lues, who were infected at least 3 years, mostly 6–15 years, before and who were between 24 and 64 years of age. It was found that twelve suffered from tabes, eleven from paresis, one from combined system disease and four others showed suspicious signs; only 22 were free from symptoms referable to the nervous system.

This selection may be dependent upon personal peculiarity and mode of life. But there are facts which speak for there being a virus from a particular source which may possess in a peculiar degree the power to produce late syphilitic symptoms. The frequent coincidence of such affections in husband and wife is a particular instance. Morel-Lavallée and Bélières have published an account of five men who were infected with syphilis from the same source and all became paretics; Bouvaist mentions that Ramadier discovered still a sixth paretic who had contracted lues from the same woman 18–20 years previously. Comparable with this is the syphilitic infection, observed by Brosius, of seven glass-blowers from a comrade, through the use, in common, of a blow-pipe. Of the five who were under observation 12 years later, one had undoubted tabes or paresis, while two others showed highly suspicious signs of these diseases. All these patients had been continually and thoroughly treated for syphilis. Of three men who were infected in one night by the same person, Nonne saw one develop tabes and two paresis. Erb also tells of five men who obtained their tabes or paresis from the same source.

It cannot, however, be lost sight of that these facts, which lend a certain credence to the theory of a syphilis which especially threatens the nervous system, cannot be extended to explain the varying susceptibility of different nationalities of people. From East Africa and Abyssinia we learn that lues has a light course and tends preferably to skin lesions while severe bone affections are rare. Düring found much tertiary lues in Asia Minor, but rarely involvement of liver, brain, spinal cord, the nerves or the eyes. But from other tropical lands we hear the reverse and further we see Europeans become paretics after infection with the same syphilis for which the nervous tissue of the natives is immune. Other influences, also, which affect all the inhabitants of the country, such as climate, must therefore be pertinent to this question. In Constantinople the different races show a very

different susceptibility for paresis, although the syphilis of the Turks, Greeks, Jews and Armenians is certainly the same.

We are forced to the opinion, then, that the difference in susceptibility to paresis must be sought, not in the different quality of the syphilitic virus, but in differences in the peoples themselves. One may consider first the habits of life which indeed have a profound effect on the peoples' health. In the first place, the situation regarding alcohol comes into question. There are not a few statistics relative to the etiological importance of alcohol; in France, especially, the principal rôle is often ascribed to it.[28] It is a known fact that lues runs an especially severe course in alcoholics; Ullman lays the blame for the occurrence of the initial lesion to alcoholic influence in 69 per cent. of the cases. Further we obtain a history of abuse of alcohol very frequently in cases of paresis. Even if we disregard the sometimes very high numbers of the Italian psychiatrists, Bär found a history of excessive alcoholism in 23.2 per cent. of his paretics, Oebecke in 43 per cent.; Hoppe, among his insane patients, established an alcoholic history in 16.1 per cent. of men and 1 per cent. of women, while in paretics the relations were 28.5 per cent. for men and 5.9 per cent. for women. The number is usually in the neighborhood of 30 per cent. The indefiniteness of the conception of the term alcoholism must, however, be considered in estimating its importance. The wide distribution of the use of alcohol in both healthy and insane people must also be referred to. With this in view the abuse of alcohol by our paretics in Munich is not of a remarkable extent; we find among the men 41.2 per cent., among the women 15.6 per cent. with an habitual daily consumption of 2 liters of beer and more. It further remains to be considered that alcoholic habits, even without excessive abuse, very frequently furnish the appropriate opportunity for luetic infec-

[28] Mairet et de Vires, de la paralysie générale, étiologie, pathogénie, traitement, 1898; Ballet, Annales médico-psychologiques, VIII, 7, 448, 464, 1898; VIII, 8, 276.

tion, and on the other hand that, often enough, an increased tendency to alcoholism is not the cause but a symptom of paresis.

From the general etiological studies, the frequency of paresis in officers, merchants, especially travellers, and railroad conductors, may here be of some use, since these people are relatively more thrown in the way of alcoholic intemperance. It is, however, much more to the point to make lues, which is common in the same classes, responsible in the first place. What appears to me more important is the difference in susceptibility of the two sexes and their variations in different countries. If the usual lesser frequency of paresis in women is to explained by their greater protection against syphilitic infection, then the relatively much greater risk in large cities goes hand in hand with the much greater tendency of women to alcoholic intemperance in these localities. Munich, with its family alcoholism, has a very strong representation of women among the paretics. The great rarity of paresis in women of higher social life is hardly to be explained by their lesser likelihood to syphilitic infection alone but may also be dependent to a certain extent upon protection against alcoholism. The ratio of women paretics in various foreign lands is worthy of special mention. Greek women are, as a rule, abstainers, while the men usually drink. In the latter paresis is common, while in women of the upper classes it is a great rarity. In one case of which I was informed by Oekonomakis, of Athens, the woman had been led into drinking habits by their husband. The Turkish women of the lower classes never use alcohol, while among the "Europeanized" women of the country, champagne has many devotees. Paresis occasionally occurs in the latter class, especially among those using alcohol, but never in the former.

Finally, it remains to be said that the peoples who are not susceptible to paresis are entirely or nearly free from alcoholic influence, either because they have no alcohol industries to flood the country with their products or because legal or religious precepts demand abstinence. As an example, the difference between

Croatians, who are susceptible to paresis, and the rarely affected Bosnians lies in the fact that among the latter as Mahommedans, as is well known, alcohol abuse is unknown. Where this customary abstinence is broken, as in the "Europeanized" Turkish and Arabian women, in the North American negroes after freedom from slavery and in the North American Indians, who are well supplied with whiskey in their reservations, there paresis makes its appearance. It is not without significance that in the previously mentioned investigation of Hoch's, there were alcoholic disturbances in 19 per cent. of the men and 6.5 per cent. of the women, while these figures among negroes were 40.8 per cent. and 8.1 per cent. Naturally, the causal significance of alcohol is not at all proven by this, but the thought is suggested that perhaps the general deterioration in the health of the race through alcoholic intemperance, still more than the personal alcoholic habit, may increase the susceptibility to paresis. This conception applies also to infantile paresis, whose relative rareness may be regarded as an indication of the harmful influence of alcohol which usually first comes in adult life.

The mental and emotional over-exertion of a life of culture is held accountable for paresis much more generally than alcohol. Krafft-Ebing has expressed his opinion of the cause of the disease in the phrase "syphilisation and civilization." The increasing struggle for existence, the haste and chase for wealth, overstraining and worry, when taken with the baleful effect of lues, allow our brains to become affected by paresis. In contrast with this are the fatalism of the Mahommedans and the idyllic states of the earlier centuries which did not subject the brain to such stimulation as does our present time.

I must confess that I cannot concur in this opinion. Under the rule of the Cæsars, in the thirty-year war and during the French Revolution, the mental lives of the great masses of people were certainly as greatly as, if not much more severely shaken

than ours at the present day and still paresis did not occur. It is difficult to see how there are any greater demands upon the brains of the Croatians than those of the Bosnians, and the Algerian paretics found by Rüdin knew no such struggle for existence as under our civilization. The same is true of the North American negroes and Indians. But even with us it is in nowise proven that paresis attacks brain workers and celebrated persons by preference. Aside from the often represented brokers who deal in millions, but who still form only a modest fraction of our paretics, a review shows only a rather large number of "men of the world" who are especially exposed to alcohol and syphilis; the great mass, however, is composed of people for whom the unrest of strife for livelihood is no greater than their neighbors'. This is especially true of the women and naturally, still more, for paretic children.

Binswanger has taken the position that we have to regard paresis as "undoubtedly the result of a functional overstrain of the central nervous system and especially of the cerebral cortex." Against this it may be stated, aside from the proven metasyphilitic nature of paresis, that those symptom-complexes which are obviously due to exhaustion do not, in any way, resemble paresis. Further, it is to be pointed out that overstrain cannot be a factor in juvenile paresis and, finally, that exhaustion, so far as we know, can produce temporary, perhaps even permanent damage, but not a progressive disease-process. It may be granted that continuous excessive emotional states, especially an unsettled life, full of ups and downs, may perhaps have a certain importance as a preparatory agent, but the relation of syphilis and alcohol is much more enlightening. Vessel injuries, to which we are also wont to ascribe a rôle in the production of arteriosclerosis, may afford an especial localization for lues. There is, however, no justification for saying that overwork, either mental or physical, has by itself any causal relation to paresis.

Why a particular syphilitic should become a paretic, we can

as little say to-day, in my opinion, as we can explain why lues one time ceases with secondary symptoms, another time with tertiary, why it sometimes attacks the liver, other times the bones or the brain. The more immediate cause for the occurrence of paresis need not lie, as will be brought out later, in the condition of the nervous tissue, but may be in very different physical peculiarities of the person concerned. We can hardly deny the importance, as a contributing cause, of the detrimental effect of alcohol which, like the paretic process, involves the nervous tissue and vessels. There must, however, be still other potent influences which render whole races subject to paresis.

Paresis does not appear, even with us, to have been associated with syphilis in earlier times. If we follow the well-grounded opinion that syphilis was brought to Europe by the followers of Columbus, then 2–3 centuries have elapsed until it has appeared frequently in its present form. One may draw an opinion that the spreading through Europe of lues has gradually generated in us those conditions which make possible the establishment of paresis. According to this conception it is still impending in the case of those nations in which syphilis has only appeared recently, as, for instance, Bosnia where, according to Glück, syphilis was introduced by the French early in the eighteenth century and where the disease in the early and frequent appearance of tertiary symptoms (30 per cent.) has the stamp of syphilis præcox. With us, also, lues, in the course of the century, has undergone marked alteration and has changed from an acute, severe disease to a comparatively mild infection. Just as in the cretin and malaria districts the condition of the whole population gradually changes, so the century-long saturation with syphilis may bring about a "reversal" in the peoples' bodies in the sense that new possibilities of injury may be opened for the syphilitic poison. It is not unlikely that this theory contains some truth. It is remarkable on the other hand with what extraordinary rapidity paresis has spread among the negroes and Indians of North

America and how relatively rare it is in South Italy and Spain, where lues is still older than in Germany.

It is also conceivable that still other changes have been fulfilled in us which have made us subject to paresis. It we consider that susceptibility to syphilitic infection is greater in the higher apes than in the lower, and still greater in man, we may understand that the whole course of development through which we have gone has evolved in us those peculiar conditions which are requisite for paresis. The training acquired in civilized life has made our brain a wonderfully capable organ, but it has also undoubtedly reduced its powers of resistance. As our domestic animals, under the protection which they enjoy, acquire accomplishments but become more delicate and non-resistant, so may also the protective qualities against paresis possessed by people of previous ages have disappeared, being still held by peoples of lower grade but showing, perhaps, a tendency to gradually become weaker from the lower to the higher apes and from them to man. In the great discussion of the lues-paresis question before the medico-psychological society in Paris, Fournier again showed that, in his experience, the apparently light cases of syphilis, *i. e.*, those without severe symptoms, were more prone to develop paresis. He cited from his practice 243 cases with severe course, none of which had become paretic, while of 83 paretics only 3 had had tertiary symptoms, 78 moderate or very slight secondaries and 2 only chancre without any subsequent symptoms. It remains to be considered whether Plaut's suggestion may not be correct, that a slight course only leads to a slight production of immunity on the part of the body—a failure in the strife to eliminate the poison. Under this viewpoint the frequency of skin symptoms in the peoples who are not susceptible to paresis appears in a clearer light.

If we bring together briefly the facts brought out in this discussion, we come to the conclusion that the distribution of paresis among peoples and classes corresponds essentially with the

frequency of syphilis among them, although the virus from certain sources seems to have especial power to produce the former disease. Further, civilized races have evidently lost the power of resistance against paresis, which is still exhibited by young people and by people living under certain conditions, possibly also to a certain extent by our rural population. This reduction of our resistance, in which possibly the long existence of syphilis among the people may play a rôle, is dependent upon the general deleterious effects of civilized life upon the race, among which influences the neglect of physical development and the weakening and poisoning by alcohol take first place.

Besides those mentioned, other determining influences for the beginning of paresis have been brought forward, especially injuries to the head.[29] Cases are, in fact, frequently observed in which the paretic symptoms make their appearance immediately or very soon after a head injury. Sometimes, as Kaplan has mentioned, the accident results on account of the paretic unsteadiness or occurs during a seizure. But, in any case, besides the paretic-like symptom picture, the evidence of syphilis can be determined by serological examination, and in this we have the true cause of the disease. Whether in head injury, as is often assumed, one has to reckon with a circumstance which favors the outbreak of paresis, cannot, in the present state of our knowledge, be either proven or argued. The same applies to heat-stroke and sunstroke which often are nothing else than paretic attacks.

Bruce and Ford Robertson[30] have recently taken an entirely new departure from the other studies of paresis. The former held paresis to be an infection by the colon bacillus from the stomach and intestine, the latter found in the mucous membrane (intestine, lungs, bladder), also in the blood of paretics, first one

[29] Mendel, Monatsschr. f. Psychiatrie, XXI, 468.

[30] Ford Robertson and Douglas McRae, Journal of Mental Science, 1907. 590.

and then a second diphtheroid bacillus ("bacillus paralyticans longus and brevis"), which he regarded as the true cause of paresis. Experimental feeding of rats with the organisms brought about nervous disturbances "like those of paresis." Syphilis is regarded, like alcohol and food rich in phosphorus, simply as a predisposing agency. Aside from the fact that Candler and Hamilton Marr in the study of numerous paretics were never able to obtain bacilli in blood or spinal fluid, the opinions voiced are so distinctly and hopelessly incompatible with all the established experience and theory regarding paresis that a detailed discussion seems superfluous.

CHAPTER V

Nature of the Disease

If we attempt now from the foregoing facts to draw at least a cursory picture of the nature of the paretic process, it must be mentioned first that we have to deal with two simultaneous but independent processes in the cortex, the destruction of nervous tissue and the vessel disease, which evidences itself in proliferation and branching as well as in lymph-sheath infiltration. To be regarded as essentially the result of these two processes are the changes in the glia to which is assigned the rôle of removing the injured nervous tissue, of filling its place and of forming a limiting reticulum. The marked fiber-increase in the most superficial cortical layer and the proliferation over the free surface indicate a more independent involvement of the glia in the disease process, if it cannot be regarded as the outcome of increased powers of growth through the dropping out of nerve-tissue. The process, so characteristically distributed in the cortex, agrees entirely in its general picture with the findings in other infectious diseases. Irritative symptoms with quick destruction or prolonged shrinking of the nerve-cell, fiber-degeneration, glia-proliferation, new formation of vessels and infiltration with lymphocytes and plasma cells are observed in a very similar manner in non purulent encephalitis, in sleeping-sickness and in rabies, which we very properly regard as due to organized toxic agents. Here we have a satisfactory agreement with the fact that paresis also is caused by an organized agent which Schaudinn has revealed to us in the spirochete pallida.

The spirochete has, to be sure, not as yet been demonstrated in the diseased tissues, in the blood or in the spinal fluid of paretics; Catola has also sought it in vain in the intestine, muscles

and lymph glands. This is true also, however, of cerebral lues and many other tertiary forms of syphilis, while the spirochete in congenital syphilis, for example, may be present in large numbers; the trypanosome Gambiense also is found in the spinal fluid in sleeping sickness. Naturally this does not show that the spirochete is never present in the body of a paretic; they may have assumed forms so far unknown to us or have located in places where we have not yet sought them or where they are hard to find. Plaut has formed this opinion chiefly on the ground of the serological observations. We know that complement-fixation is associated with the development and existence of evident disease symptoms, in other words with the life of the spirochete. It is rare in the very beginning, soon becomes very frequent, with the outbreak of secondary symptoms, recedes, returns again with relapses, disappears in 40–50 per cent. of late lues without evident symptoms and in 20 per cent. of well-treated cases, while in cerebral lues and paresis it is regularly and continuously present. Plaut has shown further that inoculation of apes with tertiary-syphilitic material does not cause the reaction to appear if spirochetæ cannot be found.

There are a number of facts which speak for paresis being nothing more than a symptomatic variety of tertiary syphilis, although occupying a rather special position. Pertinent to this question is, first, the difference in the interval between infection and outbreak of the disease. Fournier, in an instructive diagram taken from his rich experience, which is reproduced in Fig. 47, has shown, on one hand, the number of cases of brain-syphilis, on the other, of paresis, which began in the different years after the syphilitic infection. Here it is made plain that cerebral lues commences by far most frequently in the first 4–5 years, and, indeed, is quite often observed in the first and second year. After the seventh year it is much less common, but occurs in occasional cases even in the third decade after the infection. In contrast with this the first case of paresis began in the third year after the

syphilitic infection. Not until the sixth and seventh year do the cases become frequent, reaching their maximum numbers from the tenth to the twelfth year and then diminishing fairly rapidly. The last cases show an interval of 24 years.

Our experience in Germany agrees quite well with these findings. In order to secure a greater number of observations I have combined the 341 cases of Junius and Arndt with known intervals and 157 cases of my own observation, in the following table (Fig.

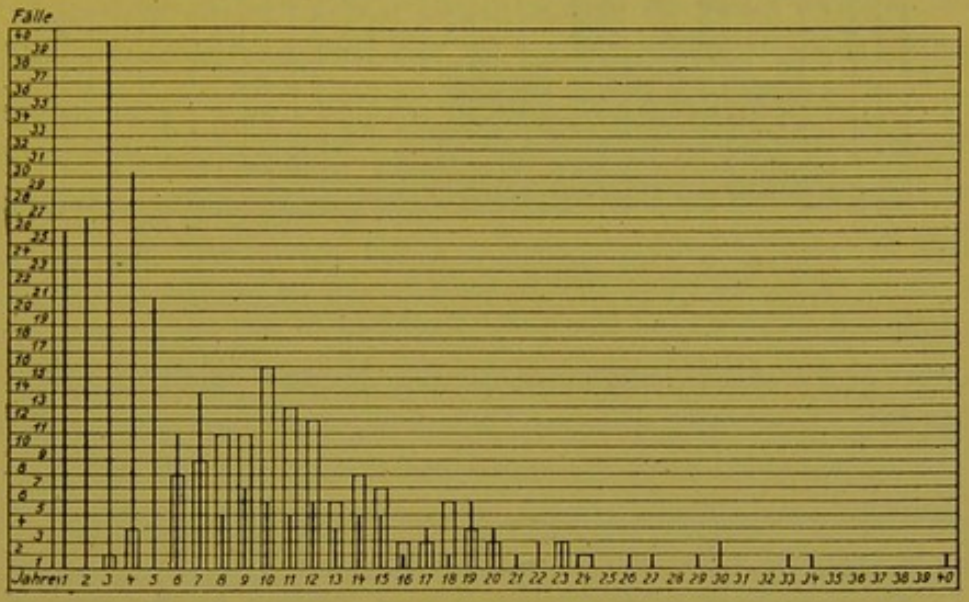


FIG. 47. Interval between syphilitic infection and onset of brain syphilis and paresis.

48). It shows that the interval between infection and the beginning of paresis is usually 10–15 years. That the tenth year predominates so greatly may be due to the tendency to give "round" numbers. The shortest interval was 3 years but only after 5–6 years do the cases become numerous. After the fifteenth year the cases fall off, at first quickly then gradually. Occasional cases are still found after an interval of 30 years and over, differing from Fournier's table probably on account of the larger number of observations. Ollivier reported a case beginning 44 years after infection.

It is worthy of note that the average duration of the interval agrees quite well with the age at which juvenile paresis usually begins, while we see the characteristic tertiary symptoms appear in the infant before or soon after birth. This furnishes a difference between the two disease forms similar to that remarked by

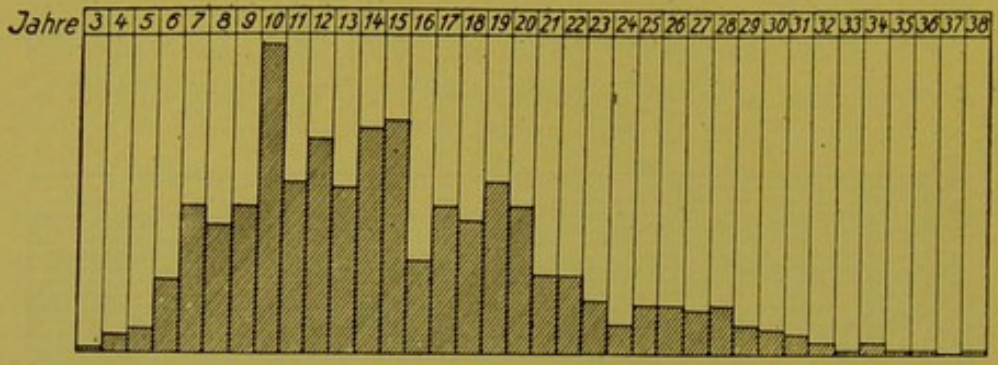


FIG. 48. Interval between syphilitic infection and paresis. (498 cases).

Fournier. Cases have also been described in persons of over 20 years as a result of congenital syphilis; Mongeri reported a similar case of 32 years of age. That such occurrences are possible cannot be denied after the experience in acquired lues, still it is naturally very difficult to positively exclude personal infection in a 20 year old individual.

Corresponding with the experience relative to the interval between syphilis and paresis, is the contribution of Heiberg who, in Copenhagen, followed continuously the number of cases of syphilis and of paresis, an observation which was possible by reason of the compulsory reporting of these diseases in Denmark. He found that 1869 and 1886 furnished an especially large number of cases of syphilis, and in 1884, fifteen years after the first date, the number of deaths from paresis in Sankt-Hans Hospital reached a high mark. Heiberg predicted a similar increase of cases of paresis after a similar lapse of time from 1886, which as a fact did occur in 1902. According to the syphilis statistics another exacerbation of paresis is to be expected in 1916 or 1917.

A second objection to the placing of paresis under the tertiary

syphilitic conditions is found in the difference in the post-mortem findings. To be sure we are not able to declare positively what are to be regarded as syphilitic changes and what not; nevertheless the peculiar anatomical findings in paresis are undeniable. Gummatous and endarteritic changes are occasionally seen, as in other forms of late syphilis, but the particular distribution of the leptomeningitis and the cortical involvement differ essentially from that of the known syphilitic brain lesions. Further differences lie in the nature and distribution of the vessel changes, the occurrence of rod-cells, the characteristic involvement of the spinal cord.

Of similar significance, although admittedly to only a limited extent, is their relative behavior to antisyphilitic treatment. Although disseminated syphilitic brain disease often is only very slightly benefited by our treatment, still we occasionally obtain a satisfactory result. In paresis, on the contrary, we not only see no improvement but the symptoms are often made worse, while the indications of late lues which occasionally accompany the disease quickly disappear.

A further difference which distinguishes both paresis and tabes from late syphilis is the complement-fixation in the spinal fluid. Since, according to Plaut, the reaction fails in paresis and tabes as seldom as it occurs in cerebrospinal syphilis, we may conclude that there are fundamental differences in the relations of the two diseases to the syphilitic process. Finally there are definite clinical differences between paresis and syphilitic brain disease in a narrower sense, which will be referred to later.

All these facts make it probable that the nature of paresis must differ in some points from that of other syphilitic mental diseases. To me the most important facts for the recognition of these differences are the late development of the disease after infection and its refractoriness to antisyphilitic treatment. The first circumstance indicates that possibly special conditions or processes must necessarily be introduced, in other words, that

some kind of connecting link must be found between the two diseases, while the latter shows us that in paresis a change has probably taken place in the original characteristics of the syphilis. These opinions are not new. Fournier has brought together paresis and several other conditions closely associated with syphilis under the term "parasyphilis" and Möbius also has referred to paresis and tabes as "metasyphilis" and regards them as sequellæ, not as immediate symptomatic forms of syphilis. Strümpell has referred to the example of diphtheritic paralysis, in which, after the first infectious stage of the disease, we see an entirely different group of disturbances appear, which we are inclined to attribute to a chemical poison originating in the disease-organisms and left behind in the system. Similarly, the organism of syphilis can, no longer, as in the beginning, through its wandering into the tissues, but through later occurring by-products of its life processes, bring about disease changes which are different in many respects from the original.

Recently research has been extended chiefly along the line of cytological and serological studies. They reveal the fact that the cell increase in the spinal fluid and the complement-fixation constitute symptoms which occur even in the very early stages of syphilis and may be continuously present. Paresis appears, then, as a disease which does not begin with even moderate abruptness but gradually prepares itself long before the actual outbreak; possibly it is already inevitable in the very early stages of lues. In this line one may suspect that possibly the continuous production of the complement-binding substance, which is regarded as syphilitic antibody, may bring about the paretic disease process.

The discovery that complement-fixation could be brought about not only with watery extract of syphilitic organs but also, though uncertainly, with certain normal body fluids and even with lecithin, further, the observation that the feces and the blood of a paretic showed an increase, the bone-marrow a decrease, in lecithin content, has led to the energetic expression, especially by

Peritz, of the opinion that in the blood of every patient there circulates a poison which may cause the destruction of lecithin and so bring about a gradual impoverishment of this important ingredient of nervous tissue. Plaut has shown that this and similar opinions may explain a prolonged, progressive illness of the syphilitic but not the process which, after a long interval, develops suddenly and within a limited space of time leads to death.

It seems to me absolutely essential for the understanding of paresis to consider that it causes, in far more aggravated form than other syphilitic affections, a severe general disturbance of nutrition of which the brain disease is the most important and most marked, but still only a part. Let us review first the numerous disturbances which paresis produces in various parts of the body in distinction from the pure brain disease. To these belong, besides the vessel-changes, the frequent disease of the heart or kidneys, which may be partly interdependent, partly due immediately to the severe general disorder of nutrition. They are certainly not brought about by the reaction of the patient, but they must have their origin in the disease-process itself. This is shown in the fact that we do not encounter changes in other organs, excluding infections, nearly so often in other forms of mental disease even if we include excitement, sleeplessness, loss of nutrition and similar conditions. The same holds for the very striking disturbances known as "trophic" and regarded as dependent upon the disease process in the nervous tissue. This explanation can only serve to a limited extent and mostly for the skin disorders. The increased brittleness of the bones is found in no purely brain affection, but occurs in tabes and various general nutritional disorders, especially in senile changes.

The excessive changes in the body-weight, which certainly are not to be explained by the patient's external conditions or habits, speak for causes which influence profoundly the general state of the body. The destruction of nervous tissue in paresis cannot, by any means, explain the enormous appetite with which the

patients are sometimes affected, the reckless satisfying of which leads to great accumulation of fat in the body, while towards the end of the disease, within a short time, the highest conceivable grade of emaciation is reached. Similar conditions are not seen in any other brain disease. On the contrary, we are forcibly reminded of the symptoms of certain metabolic disorders, especially myxedema, diabetes, chronic alcoholism. Von Noorden has shown that the immoderate deposit of fat points often to a diminished tissue-consumption in the body which is dependent upon the general disturbance of metabolism.

We have further to consider those previously mentioned observations which speak for a variety of severe changes in the condition of the blood. Also, the occasional elevation or sinking of the body-temperature, which are usually ascribed to disorder of the heat-regulating centers, may be regarded as intoxication symptoms. Corresponding disturbances in other toxic conditions are sufficiently known. One need think only of eclampsia on the one hand and myxedema on the other. Even the paretic attacks themselves can have hardly any other explanation than that of a toxic state. In the uremic convulsions, in epileptiform attacks after thyroid extirpation, in diabetic coma and in alcoholic epilepsy we have so many examples of such an origin that we may regard this opinion as most accurate. The occasional acute destruction of cells, as shown in a few cases by Nissl and regarded by Lissauer as the cause of convulsions, is best explained by a sudden overwhelming by the toxic agency of the disease. It has been justifiedly mentioned that the paretic attacks bear the stamp of localized irritative symptoms, which are difficult to account for on a toxic basis. We may, however, form the opinion from the microscopical picture that the cortical tissue is often found in quite various steps of the disease; a poison circulating in the blood may therefore produce irritative symptoms in some especially susceptible region and afterward gradually spread. The attacks are apt also to repeatedly involve the same damaged por-

tion of the cortex. One may then seek the cause of the difference between apoplectiform, general and circumscribed epileptiform convulsions in the varying relations between the strength of the toxic agent and localized susceptibility.

All the facts mentioned are, I think, only to be understood if we presume that paresis, while it disturbs the general nutrition and often involves a number of organs, vessels, heart, kidney and bones, also begets a poison which destroys large areas in the nervous system. No part of the body seems fully exempt, still there are many differences in the resistance of the different regions and cells. We find the same conditions in other intoxications, such as that of alcohol. With this agent also, different men do not always show the same psychic and nervous reaction and the symptoms do not always occur in the same sequence. Apparently the paretic poison, like the uremic and diabetic, is not present constantly nor always in large quantities in the blood stream. Rather, there may at times be an arrest in its production while at others it appears in large quantities. This, at least, is the simplest explanation of the remissions and improvements on the one hand and the convulsions and deterioration on the other. It is, therefore, not surprising that remissions as well as exacerbations of symptoms may be influenced to a certain extent by external agencies. Since the general functions of the body are seriously interfered with by the disease, it is easily to be understood that good hygienic surroundings, rest, regular life, and careful nourishment will facilitate a restoration of balance, while overwork, excesses and emotional disturbances make it more difficult. Naturally the resistance of the nervous tissue to the disease process must also in both cases be very variable.

The conception of paresis here discussed brings the disease, as one readily sees, into a certain relationship with myxedema and also with diabetes, osteomalacia and acromegaly. As the origin of this peculiar poison, which attacks the whole body at once, we may point to changes in the nature of the syphilitic

organism which lead to the production of substances of an entirely new nature. Nevertheless, so long as we have no closer knowledge of the life history of the spirochete, the opinion seems to me more reasonable, that in paresis we are dealing with a disease of metabolism which is produced in some way by syphilis in the course of a long space of time, but is not immediately related to it. Further, if we assume that in this manner the poison is elaborated, which is to be regarded as the last cause of the paretic changes, we have reached a view which, so far as I can see, best explains all the difficulties in understanding the disease.

To be sure, we can, at the present date, form no opinions as to the nature of the connecting links between lues and paresis. Mongeri found in paretics, not only frequent nephritis but also, almost without exception, a diseased liver, to which he ascribed an etiological relationship to paresis; the pernicious effect of alcohol on the liver would then explain its importance for the spreading of paresis. The bone-marrow, which is often involved in paresis, or some part of the great glandular system of our body, of whose function we know so little, may also come into question as some sort of connecting link. In this connection I may mention that myxedema, for example, may be caused by syphilis, if this disease directly involves the thyroid. The myxedema also occurs long after the syphilitic infection, since a complete destruction of the gland must precede; also, antiluetic treatment is useless, although other simultaneous indications of syphilis disappear promptly. Finally, also, the anatomical changes have no longer the slightest relation to the original syphilis.

Still closer, perhaps, is the similarity with delirium tremens and Korsakow's psychosis. Although both diseases, in their sharply defined characteristics, develop on a basis of chronic alcoholic intoxication, they have features which are essentially different from drunkenness and chronic alcoholic states, they run their independent courses even if the taking of alcohol is

stopped; in this event, to be sure, there is an effort at recovery. In delirium tremens the overwhelming with the poison appears to be only very transitory and, even in Korsakow's psychosis, only lasts for a certain time, perhaps because the primary cause, the alcohol, does not live within the body but is continually ingested anew. As the "metasyphilitic" paresis may show at the same time the anatomical and clinical signs of ordinary lues, so we encounter in Korsakow's psychosis a mixture of the immediate and intermediate alcoholic disturbances. Finally, we see disease pictures result from other allied agents, which are strikingly similar to these two diseases, namely the polyneuritic, infectious psychoses, on the one hand, and the group of pseudo-paralyses from lead, ergot, and carbon dioxide, on the other.

The especial distribution of the paretic process in the cortex has been accounted for by Mott, in the peculiarity of the blood supply, especially the course of the pial veins. Schaffer, on the other hand, has expressed the opinion that it is a disease of certain systems of nerves of especial function. Relations have been found also to the sequence of myelinization; the regions of late myelinization, the association centers of Flechsig, are attacked by preference. It is not at all improbable that we have to reckon with a varying resistance to the paretic poison in the different regions of the brain, especially in the different layers. The confinement of paresis to civilized peoples also suggests that those regions which are the seat of the highest psychic functions may be especially prone to affection. Although we know little of this matter at the present time, still the comparative cortical anatomy shows that the superficial small cell layer and especially its strong representation in the frontal region, develops latest. Further, the opinion is justified that that part of the temporal lobes which stands in close relation to speech and to abstract thought shows a stronger development with higher mental attainments. In this connection one may think first of a corelation between greater fineness of the structure as well as

function on the one hand and increased susceptibility to the paretic poison on the other. Atypical paresis, however, shows us that such relations are not infallibly present.

Edinger has recently extended his "exhaustion theory" also to paresis, in the sense that the increased demands upon the functions of the brain in civilized peoples impedes the restitution of the tissue ravaged by syphilis. Since I regard as untenable the opinion that increased strain can, in general, favor the occurrence of paresis, I cannot agree to Edinger's explanatory theory. Civilized people do not develop paresis more easily because their brains must work more, but because they injure their brains by alcohol and unhealthy modes of living, perhaps, also, because the specialized cultivation of the organ of their psychic life in the endeavor for greatest possible efficiency has rendered it more susceptible to injurious influences.

Since the final clearing up, during recent years, of its causal relation to lues and of its anatomical changes, the limitation of paresis has become much more definite. Although doubt previously existed as to how far brain-diseases of non-syphilitic origin are to be reckoned with true or "pseudoparesis," we may now regard as a clinical entity all meta-syphilitic cases, and only those, which show the previously described characteristic post mortem findings. The most recent standpoint assumed by Klippel, that the paretic symptom-complex is only a "syndrome" of very different disease processes and that, therefore, there is not one but several kinds of paresis, can as readily be ruled out as Wernicke's theory, which conceives paresis to include all the syphilitic psychoses. In our new group of paresis, all alcoholic, arterio-sclerotic, traumatic, and, in the narrower sense, syphilitic forms are excluded, while, on the other hand, certain cases of idiocy, especially progressive deterioration with epilepsy, also some cases numbered with senile dementia, prove to be true paresis. Finally we may now, with full justification, refute the constantly recurring statement that there are "transitional"

forms between paresis and many other diseases, such as senile deterioration and neurasthenia,—that they can develop one from the other. We only know that in this, as in other respects, we sometimes make wrong diagnoses; but the disease process itself, in every part of its course, is either true paresis or it is not.

CHAPTER VI

Diagnosis

The recognition of general paresis[31] is one of the most important problems of psychiatrists, because upon it depend, almost always, important legal measures, especially of a domestic nature (separations, dissolutions of business). The greatest difficulty, of course, arises at the very beginning, while the physical and mental symptoms are still indefinite. We may here apply the rule that in the case of a mental affection without obvious cause, commencing for the first time at middle life, especially in men, paresis should always be thought of. To render the case clear, it is first to be determined whether or not the patient is syphilitic and if he still bears traces of the disease. That one cannot depend upon the patient's own statement has been already emphasized. Besides, an earlier infection may be completely cured without leaving any evidences, so that it does not come into consideration in the estimation of the case.

It is therefore necessary in doubtful cases to obtain a serological examination of the blood. If complement-fixation does not occur, true paresis is to be regarded as extremely improbable. In other cases there exists, besides the possibility of paresis, that of a simple or cerebrospinal lues (if we exclude the other diseases, which give complement-fixation, but which do not figure in the practical question of differential diagnosis). One must therefore seek permission to perform a lumbar puncture. This is borne so well by paretics that this circumstance in itself may be of weight in the diagnosis. If, besides definite cell-increase, there is an increase in the content of serum-albumin in the spinal

[31] Hoche, Die Frühdiagnose der progressiven Paralyse, 2 Aufl., 1900; Heilbronner, Deutsche med. Wochenschr., 1906, 40.

fluid, a positive reaction with the Nonne-Apelt test, the findings speak strongly for paresis, still more if plasma cells but no leucocytes are found. Even if these indications fail, we cannot surely exclude paresis. A last extremely important link in the chain of proof is the test of complement-fixation in the spinal fluid. If this succeeds, the diagnosis is almost absolutely assured; one has still to reckon, to a certain extent only, with the possibility of cerebrospinal lues. When the complement-fixation does not occur here while it is present in the blood, we are dealing most probably not with paresis but with cerebrospinal, or even, in some cases, with simple lues.

Among the other somatic indications, the reflex pupillary rigidity and the peculiar speech disturbance are of greatest importance. According to Siemerling's statistics regarding pupil-rigidity in the insane, 92 per cent. of the cases were paretics; it points, in itself, only to an affection of the nervous system by lues and therefore is sometimes found in the mentally healthy and non-paretic patients, especially in tabes. It is also to be remembered that weakness of the pupillary reaction may occur temporarily from alcohol, hysterical spasm of the iris and perhaps also in psychopaths. In confused, demented or very resistive patients, especially those with restless movements of the eye-ball, it is, however, not always easy to prove conclusively the failure of light-reaction; not infrequently one gets quite different results on different days. "Sluggish" and "small range" reactions are therefore only to be used with the greatest care in the diagnosis of paresis. Pupillary difference has no diagnostic importance; irregularity of outline, on the other hand, appears to be a frequent indication and forerunner of the disturbance in reaction and is therefore important. Sometimes changes in pupil-width are brought out in different degrees of daylight where otherwise the test reveals nothing. In doubtful cases repeated examinations in the dark-room with a pupil-lamp and the influence of accommodation excluded is necessary. Still better is the employ-

ment of a measuring appliance with a magnifying lens which also allows of direct test of the other reactions, of which the secondary reaction is of especial diagnostic significance, since it seems to be absent only in paresis. Unfortunately such measurements are impossible in many patients. While the aphasic disturbances occur preferably in other brain diseases, the inability to arrange words, syllables and letters together in speech belongs, in its more marked forms, almost exclusively to paresis. The speech mannerisms of catatonic patients may resemble the slurring articulation of a paretic. If one tests the speech in the usual manner by the repetition of difficult words, we will find disturbances in many others, especially anxious or awkward patients. The speech disturbances occurring in ordinary conversation and unnoticed by the patient are therefore much more important than even pronounced difficulty with hard test-phrases.

The paretic convulsions are a very important symptom but it is, of course, necessary to exclude, from the history and physical examination, the possibility of epileptic, catatonic, alcoholic, uremic and diabetic convulsions. Transitory aphasia or quickly disappearing paralysis with or without clouding of consciousness or fainting are always very suspicious of paresis. The complete paretic attacks are characterized by their cortical-epileptic nature, the numerous localized and varying irritative symptoms and the definite but quickly compensated residuals of psychic and nervous defect symptoms. Of the other somatic signs which are of diagnostic import, the loss of tendon reflexes and the hypalgesia during inattention deserve mention; exaggeration of reflexes is only valuable when associated with ankle-clonus and perhaps with Babinski sign. Heilbronner lays emphasis on the combination of hypotonia with increased reflexes, since it points to a coexistence of posterior and lateral column disease.

The psychic symptoms which, in part, occur in similar manner in other deteriorating forms of insanity, are much less characteristic of paresis than the physical symptoms which have been

described. It is, therefore, always very hazardous to make a diagnosis of paresis solely on the mental symptoms, although there are plenty of cases in which these are very prominent while the physical signs outside of the cytological and serological findings are still insufficient to warrant an opinion of the case. The most important psychic disturbances, from a diagnostic standpoint, are early uncertainty in grasp of time relations, weakness in memory, disorder of comprehension, intelligence defect, poor judgment, dulness of mood, emotional lability and easily influenced behavior. One will do well, however, to look upon all these signs as merely sufficient to arouse a suspicion of paresis and allow them to be outweighed, in the final decision, by the results of physical examination.

In the many-sidedness of the paretic picture there is much room for failure of recognition. The patients come to the clinic with the greatest variety of diagnoses, of which I will give a small number: mania, melancholia, manic-depressive insanity, general neurasthenia, paranoia, erotic mania, delirium tremens, alcoholic deterioration, senile dementia, epilepsy, hysteria, imbecility with focal lesion, brain edema, multiple sclerosis.

Since the introduction of paresis frequently presents the symptoms of a nervous exhaustion which can be traced to some stress or worry, it is of great importance to acquire clearness regarding such cases. The occurrence of neurasthenic symptoms in a man in middle life without any particular cause is always suspicious, still it must be mentioned that a complaint of occasional giddiness, slight stuttering, tremor of the tongue and moderate increase of tendon-reflexes carry no very weighty diagnostic significance. Clear insight and appreciation of the symptoms, an effort to correct them, accessibility to judicious encouragement, progressive improvement with systematic rest, speak for neurasthenia, while the paretic is perhaps quite unconscious of obvious disturbances (memory defect, excitability) or mentions them in a hypochondriacal manner. He only incompletely understands and

heeds advice, makes remarkably little struggle against his disease in comparison with his emphatic complaints, does not follow the physician's instructions, does not keep up treatment and is often not at all improved by simple rest.

The paretic depression may show a great similarity to circular depressed states. In favor of paresis are the occurrence for the first time in middle life, the marked weakness of judgment, the mood, the behavior, and especially the memory, impaired orientation for time, loss of grasp of the surroundings, the ease with which he is influenced and to a certain extent also the senselessness and irrelevance of the delusion formation in spite of perfect mental clearness. It remains to be said that absurd time discrepancies, great defects in knowledge, inability to calculate and extreme disturbance of volition may be simple circular retardation symptoms which perhaps have disappeared on the next examination. The diagnosis from stuporous states depends on the fact that circular insanity, on the one hand, shows somewhat better comprehension and, on the other, is associated with more definite motor symptoms. The patients follow the happenings in their neighborhood with great keenness, become easily anxious if one threatens them with a pin; the inner excitement sometimes shows itself in their whispering to themselves. Stuporous paretics, on the contrary, concern themselves little with the outer world, hardly notice approaching dangers, are more free in their movements, either restless, anxious or dull and unapproachable. It is, however, naturally impossible in some cases to acquire sufficient clearness regarding the patient's mentality to differentiate quickly and satisfactorily without reference to the physical symptoms.

The expansive excitement of paresis may be confused with manic states. Aside from the physical symptoms, the inability to receive new impressions, uncertainty in recollection of dates and school knowledge (calculation), fantastic and inconsistent delusions, very striking exhibitions of emotion when talking, great

lability of mood, speak for paresis. Delusions are also not infrequently present in mania, which may closely simulate those of paresis, but one usually soon detects that the patients relate them playfully, in boasting or an attempt to frighten, not in the naïve convincing manner of the paretic. Manic patients have a certain realization of their conditions, ask frequently to go home, show a tendency to work, are not so easily quieted and reassured as paretics. In the states of severe excitement, the grasp, orientation and train of thought are much more interfered with in paretics than in manic patients. Not infrequently the history of the previous occurrence of manic or depressive attacks affords a point for the diagnosis. Often an association of manic depressive insanity with syphilitic nervous disorders occurs which may be very difficult to diagnose (reflex or complete rigidity of pupils, increase or reduction of tendon reflexes). Since here complement-fixation in the blood and cell increase in the spinal fluid may be present, the failure of complement-fixation in the spinal fluid is practically the only point for a correct diagnosis.

In the cases of acute delirium which simulate paresis the recognition depends solely upon the physical signs; at most, can the suspicion of paresis be raised by the occurrence of absurd hypochondriacal or grandiose ideas, changeable mood, continuous struggling and bellowing night and day. Inequality of facial innervation, disturbances of reflexes, especially on one side, but, above all, the reflex pupillary rigidity or the pathognomonic serological and cytological findings, will differentiate from such cases. The anamnesis may also help materially, since the paretic excited states are preceded by all sorts of introductory disturbances. The anatomical findings show, even in these cases with very stormy course, an already-existing infiltration of the lymph-sheaths (Alzheimer).

Many excited states occurring very suddenly in paretics may be confused with delirium tremens, especially if an alcoholic history exists. In general, paretics are less easily reassured, give

less information, do not show the peculiar mixture of anxiety and humor which we so often find in alcoholic delirium. Sometimes the mental weakness which follows the subsidence of the delirium is the first indication of the paretic nature of the condition, if the history and physical signs have not previously pointed to this. A true delirium tremens occasionally forms the first outbreak of the disease in a paretic of alcoholic habits.

In distinguishing paresis from the various forms of dementia præcox, certain conclusions are possible from the different sorts of mental reduction which occur. In paresis, the memory weakness, the lack of clearness and the labile mood occupy the foreground, while in dementia præcox the emotional dulness with retained memory and clearness, as well as the peculiar loss of consistence between ideation, emotion and will are the salient features. In the paretic dementia, although refusal of food, dulness and failure to react may be present for a long time, we do not encounter the perversity, mannerisms, periodic excitement, rigid stupor and stubborn negativism of dementia præcox. During excitement we observe some stereotypies, but not the irrelevant replies and jumbled speech; the patients are not entirely conscious and mentally clear as catatonics usually are. These last mentioned points together with the pliability, the peculiar happy mood, the rarity of auditory hallucinations and ideas of bodily influence, finally the whole development of the disease distinguish paresis from dementia phantastica with its abundant but often paretic-like delusions.

By far the most difficult is the differentiation of paresis from those diseases which also show extensive cortical disturbances. Among these are the syphilitic brain diseases and arteriosclerosis of the brain; sometimes, also, certain forms of senile dementia. Between paresis and syphilitic pseudo-paresis the chief difference is that, in the latter, the mental disturbance is far less, in comparison with the physical. Moreover, the psychic disturbances are usually of an irregular character, causing a

profound disorder in one sphere or function while another is left intact. This has led the French to speak of the syphilitic dementia as "demence lacunaire"; that of paresis as "demence globaire." Patients with pseudo-paresis are apt to possess clear insight throughout. Many retain orientation and grasp and are always neat and orderly while a paretic with severe physical symptoms would be most unlikely to have a similar retention of his faculties. In the physical disturbances there is likewise a focal tendency. While in paresis, as the disease progresses, all the characteristic signs of disordered pupils, reflexes, speech and writing, paralysis and contractures appear eventually; in pseudo-paresis these show great variations. The speech and writing are usually but little disturbed and are often normal. Speech disorder when present is more ataxic or paretic than apractic, *i. e.*, it may be slurring and indistinct without distortion in the arrangement of words and syllables. The pupils in pseudo-paresis may be constantly normal; unilateral symptoms with ankle clonus and Babinski sign are common. The eye-muscle paralyses play a much greater rôle than in paresis. The insensibility to pain when attention is distracted which is so characteristic of paresis seems to fail or to be very slightly evident in pseudo-paresis. The courses of the two diseases also show important differences. The development of pseudo-paresis is, in general, much more prolonged and irregular. After a severe seizure with subsequent paralysis, a period of arrest of all symptoms may occur, lasting for years. As a final point, pseudo-paresis leads to death only in the severe cases and is not regarded, like paresis, as certainly fatal. Usually the patients reach a state of slight or severe chronic illness with a mixture of mental and physical symptoms which continues indefinitely without change.

In differentiating paresis from arteriosclerotic insanity an important, though not always useful, point is the greater average age of the patients in the latter condition. Then it is to be remembered that paresis, in both its mental and physical characteristics,

is a diffuse process, while in arteriosclerotic psychosis the symptoms are those of numerous, usually small focal lesions. In the former the mental symptoms are much more prominent by comparison with the physical and are usually in evidence long before the somatic changes attract attention, while in the latter the paralytic stroke is very often the point of outset for the psychic symptoms. Fatiguability is a much more prominent symptom than in paresis, possibly on account of nutritional disturbances in the brain resulting from arteriosclerosis. In arteriosclerotic patients the disturbance of retention is much greater than that of memory, while in paresis they are equally prominent or memory is more affected. The emotional coloring in paresis is greater and the extreme feeling of happiness of the paretic is never seen in the arteriosclerotic condition. In the latter the delusions are far more sensible and uniform, and grandiose ideas are rare. Insight and feeling of illness are usually present in arteriosclerotic cases, in contrast with the carelessness and indifference of the paretic. The physical symptoms of arteriosclerosis are more severe and permanent than in paresis. Circumscribed paralyses with spasms, aphasia, word-deafness, hemianopsia and other focal signs are common. The speech of arteriosclerosis is seldom ataxic, the writing shows omissions and paragraphic disturbances rather than ataxia or distortion; perseveration is very prominent. The pupils show only diminished reaction. I doubt very much if reflex pupillary rigidity, except possibly in extremely rare instances, ever occurs in other than syphilitic and metasyphilitic conditions.

In the differential diagnosis from Korsakow's psychosis, the history is of great weight; in the one we have alcohol, in the other syphilis; in Korsakow's disease we have the retention more disturbed, in paresis, the memory. The prominent neuritic signs speak for Korsakow's psychosis, also the eye-muscle palsies and fine tremors. On the mental side the dulness or characteristic alcoholic humor are to be placed against the happy, demented

condition of the paretic. The memory defect of the paretic is not circumscribed as in the alcoholic psychosis but involves events of youthful years as greatly as more recent happenings.

In all these last three conditions, especially in the arteriosclerotic and Korsakow's psychoses, the complement-fixation test is of the greatest importance.

Juvenile paresis may have to be diagnosed from idiocy, as is often the case in the congenitally syphilitic imbeciles. Differentiation depends upon the pupillary and reflex disturbances, the complement-fixation test and the progressive course. Occasionally brain tumor produces a picture similar to paresis when the cranial pressure causes a simple general mental reduction, without localizing symptoms. Choked disk, stupor and impaired consciousness without great disturbance of memory and intelligence, as well as severe headaches, speak for tumor; ataxic speech, pupillary rigidity and complement-fixation are in favor of paresis. In the rare cases in which brain tumor develops in paresis (gumma, glioma) one finds both lines of symptoms occurring simultaneously, while in paresis with permanent focal symptoms, the characteristic general dementia, the behavior of the pupils, the speech defect, the absence of choke disk and finally the complement-fixation in the spinal fluid render a correct diagnosis possible.

Finally, it is still to be mentioned that there are, especially in the involution period, all sorts of disease processes which are as yet but little understood and which, because of their producing mental weakness and partly because of the physical symptoms accompanying (seizures, slight speech disturbance), may sometimes be confused with paresis. Of the means of differentiation of paresis, which we have acquired in late years, the results of cytological and serological tests are of greatest importance in the diagnosis.

CHAPTER VII

Treatment

The combating of paresis must begin with prevention. In the first place, all measures must be taken which tend to work against the spread of lues; their instigation, testing and carrying out we must naturally leave to the syphilologists. It need only be mentioned that the campaign against alcohol, which comes within our province, assists materially in restricting lues, since it is a matter of experience that a very considerable proportion of infections occur under alcoholic influence. Further, in view of the unfavorable influence of alcohol upon syphilis, we must caution very urgently against the use of spirituous liquors, those who have had the misfortune to become infected. Constant observation of the blood and spinal fluid is important. Although we are by no means in a position to say that the disappearance of pleocytosis and complement-fixation mean the cure of the syphilis, we can nevertheless see in those symptoms an indication of a menace to the nervous tissue. We should then strive to overcome them. The lymphocytosis is doubtlessly influenced by antisyphilitic treatment and this is true to a lesser extent of complement-fixation.

Fournier has laid strong emphasis on his opinion that by systematic and thorough treatment of syphilis, paresis may be prevented. He attributes his previously mentioned experience regarding the fatal course of the apparently light cases of syphilis to the circumstance that such cases seldom or never receive sufficient treatment. According to his reckoning only 5 per cent. of paretics have gone through a course of treatment sufficient for all requirements. He therefore advises in the first two years after infection a thorough course of mercury which should be repeated in the fifth, seventh and eighth years and even longer. Most

other writers upon the preventive action of mercurial treatment are much less convinced. Kiss found that 77.1 per cent. of his paretics had had antisyphilitic treatment; Schuster, of whose patients 17–19 per cent. had had several courses of treatment, thinks that no influence either upon the frequency of paresis or upon the complement-fixation can be demonstrated. Of the Dalldorf patients (Junius and Arndt) 45.2 per cent. had had antisyphilitic treatment, often only locally. Marcus found among his paretics that the treatment had been but partly carried out and I may also say that in the great majority of the cases one learns of only a single course of inunctions or injections, often enough of only one dose. Hudovering and Guszmann determined that among 28 cases of metasyphilitic and late syphilitic brain and cord affections, a half had received no treatment and only five a sufficient amount. It will require new and extended experience to settle definitely the question of the preventive value of an early and thorough treatment of syphilis. Since, however, the development of paresis is not favored by mercurial treatment one may, from a psychiatric viewpoint, strongly advise its carrying out, especially if the examination of the blood and spinal fluid point to a continuation of the syphilitic process.

It is quite a different matter if the paresis has already broken out. Even here, on account of its known syphilitic origin, the attempt is being constantly made to influence its course by antisyphilitic treatment. Experience has shown that the results from mercury and potassium iodide are even less than in tabes. Remissions sometimes occur, just as they do with other forms of treatment, especially under the influence of hospital rest. Räcke reported that of 28 cases in which antiluetic treatment had been instituted, improvement occurred twelve times. Wagner advises the use of mercury and iodide in conjunction with thyreodin. Marchand secured improvement in four of seven cases by the injection into the spinal cord of mercury biniodide (0.002 g. with potassium iodide 0.02 g.) but this drug is not without its dangers.

On the other hand, in a number of cases in which syphilis had certainly preceded, and in some with fresh signs of the disease, I have seen a rapid falling off of strength and sudden onset of acute excitement immediately following a course of inunctions; Buchholz also saw two cases of galloping paresis start this way. I can therefore, in agreement with the majority of psychiatrists, only advise that, for the present, one should be content with potassium iodide. Only if obvious signs of syphilis are present or, in doubtful cases, if there is a possibility that we are perhaps dealing with simple luetic brain disease, will all doubt regarding the advisability of a course of mercury be removed.

The similarity of paresis to sleeping-sickness has led to the employment of atoxyl, which is useful in the latter disease, but unfortunately without result. After Ehrlich succeeded in producing the still more active agent against sleeping-sickness, arsenophenylglycin, this also was used by Alt in paretics. The susceptibility of the patient was first tested by injecting a 10 per cent. solution into the tissues and rubbing it into the skin; then, on two successive days, 1 gram was injected into the muscles. The reaction symptoms observed were elevation of temperature, acceleration of pulse, nausea and vomiting. In 7 of the 31 paretics so treated the complement-fixation disappeared, only to return again in five weeks; once a reduction of lecithin in the stools was observed, another time in the blood. Whether with this or the newest preparation of Ehrlich ("606") we can bring about recoveries in paresis, must be left to the future.

The occasional occurrence of marked improvement and even apparent recovery of paresis after prolonged purulent affections has led to numerous attempts to produce such conditions artificially and thus benefit the disease. Earlier this was accomplished by tartar emetic salve or even by the introduction of a piece of bone to rub upon the skull, a procedure which, especially in restless patients, was associated with grave danger. Recently the experiment has again been taken up by the Vienna

school[32] in the shape of injecting tuberculin in increasing doses (0.01–0.1) and over a prolonged period. The following out of 66 cases so treated appears to show a slightly favorable influence. In the first year 20 of these cases died, against 39 of a like number of untreated cases; after three years, eight of the first group were still alive, of the latter, only five, of which one was in a five year remission. Since, also, after the experience of syphilologists, the introduction of the organisms of septic disease hinders the propagation of spirochetæ, the possibility of securing, in bacterial toxins, an ally against paresis does not seem to be excluded. Wagner has recently again gone in extensively for these experiments. Fischer, with the idea that the leucocytosis accompanying infections may have a favorable influence, injected his paretics every three to five days with ½ gram of the sodium salt of nucleinic acid in 10 per cent. solution, which quickly produced a marked increase of leucocytes. He reports that four of 22 cases so treated showed pronounced remissions, while no such change was noted in any of the control cases. However, the procedure must be continued over a long period before a final judgment regarding its efficacy can be reached. Juvenile paresis, the longer duration of which would perhaps allow more latitude for the elaboration of protective mechanisms, should be particularly suitable for this.

In his special contribution to the nature of the paretic disease process, Peritz lends his support to the proposition to treat paresis by injections of lecithin; in some cases he saw the disappearance of complement-fixation result therefrom. Robertson and McRae employ suspensions of their cultures of "bacillus paralyticans" and obtain good results by rubbing into the thighs of paretics and tabetics. They also obtained an anti-serum against paresis by inoculating sheep, and are of the opinion that not only is a diagnostic febrile reaction produced but also a curative effect. Bruce recommended the injection of a serum from a

[32] Pilcz, Jahrb. f. Psychiatrie, XXV, 141.

paretic in a remission, Lechner the serum of horses treated with paretic serum.

Since the treatment of paresis based on its etiology is not yet out of the experimental stage, we are forced chiefly to combat its symptoms. The most important measures in this direction are rest, isolation from the usual surroundings and business as well as a careful regulation of the general mode of life. Excited patients and those with suicidal tendencies should, without question, be kept in an institution in order to protect themselves and others from their acts. Quiet and tractable patients in better circumstances may be kept in private home care so far as intelligent treatment and watching are possible. Bathing resorts, however, with their many distractions and excitements, tiring journeys, weakening regime, exhausting cold-water baths, etc., should be forbidden. It is a common experience to observe a rapid deterioration in the general condition and a sudden outbreak of excitement as a result of the abuse of cold baths. Visits to the seashore, as a rule, are not favorable; cold sea bathing should be strongly advised against. Besides rest, careful nourishment, regulation of digestion, fresh air, abstinence from alcoholic liquors, tobacco, coffee and tea are important. A very mild well-supervised hydropathic treatment (rubbing, warm baths, packs; no douches or showers) may be of benefit. Alt has suggested that the most advantageous composition of the diet had best be determined by tests of metabolism.

In the excited states of paretics, the transference to quiet surroundings and rest in bed very frequently help, as well as a bath and friendly and careful diversion, in keeping with the patient's mood. If the excitement is very great, treatment in the continuous bath is indicated, perhaps first with the assistance of veronal or hyoscine, later without drugs. As the patients often do not take sufficient nourishment, it is sometimes necessary to resort to tube-feeding once or twice daily.

The greatest difficulty is met with in the treatment of the

anxious excitements of paretics. Here, baths and packs are sometimes impossible and sedatives have little effect. One is therefore often limited to continuous watching, protection of the patient from injury and careful treatment of abrasions which may occur. Nevertheless it is often impossible to prevent the occurrence of furuncles and cellulitis. I have occasionally tried to cause improvement in marked, senseless excitement by systematic subcutaneous infusions repeated twice daily; we introduced each time 750 c.c. of saline solution. The treatment was continued, in one case, for two weeks without bad effect. The patient, who seemed to be doomed to a rapid decline, underwent marked and lasting improvement, so that further use of this procedure in such cases may be warranted. Donath has recently recommended very warmly the systematic employment of infusions in paresis; he uses, besides 0.85 per cent. saline solution, a complex isotonic solution.

Paretics in the last bed-ridden stage require the most care, especially in the attacks. Even before this, it is often necessary to attend carefully to the cleanliness of the patient and to watch the taking of food, also, on account of the careless chewing, to give only finely cut, easily masticated food and to prevent the greedy gulping of a meal, since a fatal suffocation can easily occur. During convulsions and in very demented patients the prevention of bed-sores is of the greatest importance. Sometimes the best means to this end is the continuous bath with the patient lying on a stretched sheet or possibly on a water-bed. If this is not practicable it is of most help to maintain strict cleanliness of the threatened part by frequent washing with cold water or solution of bichloride in alcohol, careful removal of wrinkles, bread-crumbs, etc., from the bed, the use of water or air pillows or lying upon cotton-wool or moss which quickly absorb urine or other moisture; unfortunately these are often torn up by demented patients. Finally, a regular changing of the position of the patient by an attendant is necessary so that the patient (in

severe cases every ½ hour, day and night) lies alternately on his side, abdomen, back, etc. This method, devised by von Gudden, which also, to a certain extent, obviates hypostatic pneumonia, has, for decades, made it possible to almost do away with the otherwise inevitable bed-sores of paretics (10 per cent. die of this cause, according to Mendel) and, in any case, the very dangerous forms are prevented. It is much more difficult to cause the bed-sore to heal when, through insufficient care or neglect, even for a few hours, it has once become started. Since the patients, by their restlessness and tearing the dressings, often render the usual surgical treatment very difficult, such cases make the greatest demands upon the patience and attention of physicians and nurses. Since the employment of the continuous bath by day and night, even such cases have lost most of their dread. The patients take very kindly to the treatment and the sores heal, with occasional surgical help, without any complications.

For the treatment of paretic seizures Kemmler advocates packing the head in ice, in severe convulsions enemata of amyl hydrate (6 g.); this drug may also be given subcutaneously in 5–10 per cent. solution. If a quick effect is necessary, chloroform narcosis to the point of quieting the motor symptoms may be resorted to. In cardiac failure stimulants are indicated, such as caffeine, camphor, alcohol in small doses, but especially saline infusions and inhalation of oxygen.

The emptying of bowels and bladder requires assistance by enema or catheterization usually only in the beginning of an attack; later the functions operate by themselves, although too long a delay may allow of overfilling and resulting paralysis of both organs, after which they will require constant artificial means. Unfortunately the treatment of vesical paralysis is often interfered with by stricture. It is useful to follow catheterization by washing out the bladder (boric acid), which may be given at a cool temperature when there is laxness of the sphincter. At other times than during attacks the evacuation of urine and

feces also needs constant attention if one would avoid urine dribbling and loss of control of the rectum. The physician should accustom himself to note, by palpation above the symphysis, the fulness of the bladder in his paralyzed or bed-ridden paretics on each of his visits. Often a warm bath at the right time will stimulate a voidance. Vesical inflammation requires irrigation and urotropin. In one paretic who had already been catheterized for two years, independent micturition was restored by four weeks of irrigation (tannin solution) twice daily, nor was it lost during a paretic attack lasting 13 days. In this same patient, with the above-mentioned treatment, no bed-sores occurred up to the day of death, in spite of deep coma and almost complete pulselessness. Nourishment should always be given by tube during attacks (necessary only in attacks lasting many days); simply pouring food into the mouth is highly dangerous. If one is careful to clean and disinfect the mouth frequently with a moist cloth (potassium chlorate) and to maintain the moisture of the cornea by regular half-hourly moving of the partly-opened eye-lids (to prevent ulceration), one will often succeed in keeping the patient alive even through an attack lasting 8–14 days.

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