The general progressive paralysis of the insane (dementia paralytica)

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Gentlemen! The form of illness that we want to deal with in more detail today is one that I have already brought into the circle of our clinical discussions several times when it was a matter of making differential diagnostic considerations. You will remember that in the relatively simpler clinical pictures of mania, melancholia and hy- pochondria, especially when they were diseases of middle age, as well as in the more complicated mental diseases, which belong to the etiologic-clinical group of exhaustion, intoxication and infection, Intoxication and infectious psychoses, have repeatedly discussed the question whether the mask of a functional psychosis does not conceal that serious organic disease of the central nervous system, the fatal significance of which is now generally recognized not only from a medical but also from a social point of view. Every layperson knows that the medical diagnosis of dementia paralytica, "softening of the brain" [1]is popularly considered to be tantamount to pronouncing a death sentence on the unfortunate patient.

Even more urgent and immediate was the need to include dementia paralytica in our diagnostic considerations in all those mental disorders which showed clear symptoms of mental and somatic deficits, thus documenting their affiliation with organic brain diseases. Here again, certain forms of infectious and intoxication psychoses, especially those caused b y syphilis and alcohol, came to the fore, as well as those which develop on the basis of arteriosclerosis, primarily on the border between manhood and old age. I have endeavored to show you again and again what a changeable and prognostically deceptive picture the initial stages of dementia paralytica in particular can present. This makes it all the more compelling to present this disease to you in its various stages and manifestations in a large number of cases and to emphasize from the almost overwhelming abundance and variety of symptoms those which give the disease its specific character and establish its diagnosis. Above all, it is my task to give you a summary of the etiologic-clinical and clinical-symptomatologic aspects which enable us to differentiate this disease from related brain and spinal cord disorders.

Dementia paralytica was not recognized until the second decade of the 19th century.

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[1]This anatomically incorrect and misleading term has become so firmly established in all sections of the population, especially in the most educated circles, that all efforts on the part of scientific circles to eradicate this term must be regarded as unsuccessful.

was recognized as an independent disease. It was Bayle who first recognized that the progressive symptoms of paralysis belonged to a clinically peculiar mental disorder, the final stage of which was mental atrophy. He came to the conclusion that both series of disease processes were based on an organic disease process of the brain, which he interpreted as arachnitis chronica. Over the course of the next decades, this finding was retained in clinical terms despite manifold challenges and further developed in pathological-anatomical terms. By the 1950s, knowledge of dementia paralytica had progressed to such an extent that Falret and others were able to attempt to distinguish it from related non-genuine forms of simple insanity with paralysis and also to separate it pathologically and anatomically from other organic diseases of the brain. For years, researchers have endeavored to trace dementia paralytica back to a unified scheme encompassing all disease processes, both anatomically and clinically. The same pathological-anatomical cause should correspond to the same or at least only within certain narrow limits fluctuating disease symptoms. In this way, a typical paralysis with a certain course of the mental disorder was constructed, which one tried to base on certain pathological-anatomical foundations. However, the further the psychiatrists' research extended in both directions, the clearer it became that this disorder may occupy a much larger space than the original sketch would have indicated. It was seen that the range of mental symptoms was much greater than was at first supposed, and the view was finally arrived at that de mentia paralytica, especially in its first stages, could simulate all kinds of functional and organic nervous and mental disorders. It was gradually established that, given the diversity and colorful alternation of mental illness symptoms, it is impossible to construct certain clinical disease types by utilizing the predominant mental illness symptoms. All attempts to find in this way not only lawful phases of the course, but even certain forms of disease, must be regarded as having failed. The old typical paralysis with the lawful succession of a) the stage initiale with incompletely developed symptoms, b) the stage melancholicum
s. hypochondriacum, c) the stage maniacale, d) the stage dementiae is only apt to mislead the beginner and to make the most essential of mental disease processes, namely the deterioration of intelligence, appear to him as the final link in a long chain of disease processes. It is equally erroneous to want to establish a series of other sharply defined mental clinical pictures alongside this typical form of illness. If you survey the literature, you will encounter the most diverse attempts of this kind. It is sufficient to enumerate a number of such varieties, the agitated, the depressive, the simple demented, the circular, the stuporous, the catatonic, the hallucinatory form, to show you the fragmentation to which these attempts have led. I beg you to note that all

these tests suffer from the same deficiency: The clinical diagnosis is based on extremely variable variables and other, much more significant, because more constant symptoms are pushed into the background.

On the basis of aetiological-clinical and anatomical considerations, it is justified to assume that dementia paralytica is one of those diseases for which our present conditions of existence provide an extraordinarily favorable breeding ground. The devastating influence of present-day cul- tural and social harmfulness is felt first and foremost by those individuals whose central nervous system can offer the least resistance to the highly increased demands on its performance. This inadequate resistance can be innate or acquired. In the former case, it is expressed either in a morphologically detectable inhibition of development or, to a lesser degree, in a functional weakening of individual or all organ systems. It is undeniable that these constitutional weaknesses lead mainly to damage to the central nervous system given today's demands on nervous performance. This also explains the general increase in nervous and mental illnesses in general. The weakening of the nervous capacity acquired during an individual's life is primarily caused by the effect of harmful factors which have an unfavorable influence on the nutritive processes within the central nervous substance, reduce work performance and ultimately lead to more serious destructive disease processes. If both series of these pathological developmental conditions work together, the devastating influence of the harmful effects of culture will be all the greater. The two main types of this nervous culture disease are neurasthenia in the field of functional nervous diseases and dementia paralytica in the field of organic diseases of the central nervous system. Both diseases have many points of contact with each other.

Dementia paralytica comprises a series of clinical pictures that vary greatly both in terms of the number and grouping of symptoms in the mental and somatic areas and in terms of the time course of the symptoms. What they all have in common is the inexorable progression of mental and physical deterioration and the fatal outcome. In the interest of a clear presentation of this disease, it is necessary to divide it into four stages according to its development and course:
a) the prodromal stage (also called the neurasthenic stage), b) the initial stage, c) the acmes stage, d) the terminal stage. The task of the clinical presentation is now to describe the overall symptomatology (the psychic and somatic disorders taken together) within these stages and, on the basis of a precise analysis, to illuminate the peculiarities of each individual case. We shall see later that in this way it will be possible to distinguish individual groups of cases of illness which are characterized by the combination of certain symptoms in the psychic and somatic fields and, above all, by the similarity of the course of the illness.

proved to belong together to be more clearly separated from each other. These attempts are further supported by the fact that each individual subtype of typical paralysis also appears to have a pathological-anatomical peculiarity.

1. Prodromal stage.

In the vast majority of cases, the symptoms of the precursor stage of the disease can be traced back many years. You remember the patient I recently introduced to you. He was a 34-year-old man with a weak constitution and pronounced kyphoscoliosis. He had contracted syphilitic infection 10 years ago. At that time only slight secondary symptoms appeared, which quickly and finally disappeared after a course of injections. Four years later he fell ill with the pronounced symptoms of neurasthenia. He suffered from insomnia, head pressure, mental and physical fatigue, indigestion, palpitations and dizziness. A cold water cure and several months away from his office (he is a court clerk) apparently restored him completely. He married a year later. The marriage produced two healthy children. He worked in his profession without any disturbance to the complete satisfaction of his superiors. One year ago the neurasthenic symptoms appeared in the same way as before and increased. The patient continued to work, however, until three months ago, when further conspicuous symptoms were added which made his admission to the clinic necessary. At that time, the case was considered by the attending physicians to be neurasthenia and the diagnosis of progressive paralysis was only established by clinical examination. We will encounter this observation again later when we discuss the second stage of the condition, as this case is a useful example of "typical" paralysis. Here we are only interested in the neurasthenic antecedent symptoms, which were demonstrably present six years ago. How are they to be interpreted and how do they relate to the later onset of the disease? From a pathogenetic point of view, neurasthenia is the neurosis of exhaustion κατ' εξοχήν, which either affects neuropathically predisposed individuals or, in the case of a completely normal mental and physical disposition and development, is caused exclusively by the harmful factors that affect people during their individual lives. The most important of these harmful effects are Violent, prolonged emotional excitement, physical and mental overexertion, intoxication (alcohol, morphine, lead, arsenic, etc.), infections (influenza, typhoid, malaria, syphilis, etc.). It goes without saying that all these harmful substances become more disastrous and cause neurasthenia more easily and more intensely when they affect neuropathically predisposed individuals. As experience teaches, in many constitutional weaklings even relatively minor harmfulness or average physical and mental performance is sufficient to bring about the breakdown of the nervous power balance. In the case mentioned above, the etiologic conditions that impaired the patient's physical development have not been fully elucidated; his mental development is said to have been completely normal. The main damaging cause here is certainly the

syphilitic infestation. With this statement we touch upon the most burning question of etiologic-clinical paralysis research, but at the same time also the difficulties of differential diagnosis between neurasthenia and paralysis. Here we have to consider mainly the following experiences: The syphilitic infection creates, apart from all the local manifestations of disease which result from the specific effects of the syphilitic poison on the individual organs, other disorders whose pathogenetic significance still needs to be clarified, but which have long been known clinically. They appear to us either as disorders of general nutrition, which can lead to pronounced cachectic conditions, or as functional weaknesses that only affect individual organs or organ systems. The nervous disorders that arise on this basis used to be called syphilitic hypochondria, an expression that has now given way to the more common term "syphilitic neurasthenia".

You know that, for practical reasons, I am advocating the absolute fusion of these two symptoms and that the old term hypochondria is more appropriate for many cases of so-called syphilitic neurasthenia, since severe affective disorders and hypochondriacal delusions are quite frequently in the foreground as psychological symptoms. As you will see later, syphilis is also the most important Quelle of the destructive disease of the central nervous system that underlies paralysis. Given this similarity in the cause of the disease, are there closer connections between syphilitic hypochondria and progressive paralysis, or are the pathological processes underlying both diseases fundamentally different? I would like to answer the former question in the affirmative, for the following reasons. I am convinced that there is no fundamental qualitative difference between the damage to the nervous substance which underlies the functional and that which underlies the organic nervous and mental diseases. Functional neuroses and psychoses are based, as I have already explained in more detail elsewhere, primarily on nutritive disorders of the function-bearing nerve substance and in particular of the central nerve cell. These nutritive disorders can be based on damage to both the assimilatory and dissimilatory processes within the nerve cell. In an attempt to link the more recent research on the histology of the central nerve cell with these pathophysiological definitions, I came to the conclusion that the compensable functional disorders are based on partial damage to the nerve cell, which is easily accessible to complete regeneration by the physiological metabolic processes. The lightest degrees of this, which come closest to fatigue, are most probably due to partial damage to those components of the nerve cell that are stacked in the Nissl bodies. The more extensive this type of molecular damage is, the more difficult and protracted it will be to replace and rebuild these cell components. It will also be more difficult and imperfect in all those individuals in whom, if I may say so, the molecular constitution of the nerve cell was already inferior before the damaging causes came into play. But even then the replacement will be an inadequate and imperfect one, if the relation between as- similation and dissimilation (supply of force, consumption of force, and replacement of force) has been altered by the persistent action of the damaging causes.

The central nervous system is disturbed for a longer period of time by increased demands on it or by a reduction or respect- ive deterioration of the nutrient material supplied to the cell. There will thus be smooth transitions between the completely compensable and the permanent functional disorders, which have found their clinical expression in the concepts of permanent fatigue and exhaustion. Whether these latter disorders are also based exclusively on damage to the *nervous system* or whether it is not rather the function-bearing nerve substance in the narrower sense, the neuro-soma Hehls , that is affected, must remain an open question. In any case, even in this second case, in which the nervous performance is reduced but not eliminated, or rather permanently destroyed, it can only be a matter of tartial damage. In the cases that interest us here, the syphilis toxin is to be regarded as the damaging effect, and according to the more recent views it is most probably a matter of toxins acting on the nerve substance, which have been produced by the metabolic processes of the hypothetical organized carrier of syphilis and supplied to the tissue fluids.

If these toxin effects are of a more intensive nature and if they occur together with other processes that impair the nutritional conditions and performance of the nerve cell, their influence is all the more pernicious. The disease process does not remain at the stage of partial damage, rather it gradually takes hold of the entire nerve cell. The partial damage becomes a total disease, which ends in the destruction not only of the *Nissl* granules, but also of the function-bearing substance in the narrower sense. I have been able to prove to you that these deductions also have a scientific justification today through histological demonstrations of diseased nerve cells of the cerebral cortex and spinal cord, which we have found in two cases of delirium acutum on an infectious basis (influenza and varicella). They were able to precisely follow all stages of the disease process in these acute brain diseases with severe mental disorders, from partial damage to the nerve *cells* to their complete destruction.

What do these findings teach us about the pathogenetic relationships between syphilis, neurasthenia and paralysis? First of all, it must be emphasized here that, in contrast to the stormy course of delirium acutum, the pathological processes in the syphilitic diseases mentioned take place much more slowly and sluggishly. The syphilistoxins appear to develop only to a lesser extent for reasons that are as yet unexplained and also, as far as can be concluded from clinical facts, have a far weaker mode of action. In a large number of cases, the damage remains at an incomplete stage of development and then manifests itself either in compensable or uncompensable functional disorders, which clinically and symptomatologically encompass the most diverse clinical pictures, from simple neurasthenia to complicated cases of paranoia, and are only held together conceptually by the common cause. It has not yet been possible to prove the anatomical changes in the nervous substance which underlie these cases. However, as you know, I am inclined to the view that precise histologic examinations of the central nervous system and the peripheral (visceral-sympathetic) nerves can also be used here, if the patients are affected as a result of sy-

I have been able to demonstrate degenerative processes in the fibers of the visceral nerve plexus in at least two cases of severe visceral syphilis with severe anxiety and fatal outcome (by suicide).

These pathogenetic disputes, which are already in the anatomical field, were essential in order to make clear to you the intimate connections between the so-called functional and the organically caused syphilitic diseases of the central nervous system. They showed us that it is not possible to draw a sharp line between the two, as they are not qualitative but quantitative differences in pathological processes. Indeed, the question is made even more complex by the fact that the long-lasting, probably intermittent, damaging effect of syphilis toxins on the central nervous system can lead to partial damage with compensable disturbances of nerve function at some sites and to uncompensable disturbances at other sites, while total damage with loss of function can occur at third sites. The clinically significant cases, which are so difficult to diagnose, then arise in which, in addition to certain permanent focal symptoms, only the clinical features of a more or less pronounced neurasthenia are present. I may only remind you here of the cases to which I have already repeatedly drawn your attention, in which pronounced reflective pupillary rigidity is present in neurasthenic individuals who have previously suffered from syphilitic disease, without the paralytic disease process being added even after years of existence of this clinical picture. So you see that organic lesions in the area of the light reflex center can combine with neurasthenic symptoms without the latter having to be the ominous harbingers of a general organic disease of the central nervous system. I have pointed out to you the rarity of such observations and have informed you that in the vast majority of cases such findings are partial manifestations of tabes or dementia paralytica. The same applies to the early appearance of *Westphal's* sign. After syphilitic infestation, it may persist in rare cases as the only symptom of failure and combine with syphilitic neurasthenia. In such cases, too, we shall always have the most urgent suspicion of the subsequent development of tabes or paralysis, but we shall not be justified in making a positive diagnosis of the development of either of these diseases, or of both taken together, until the other evident features of these diseases have been added. In such cases we must exercise the greatest caution; the most exact control of the case, which must extend over a period of years, is necessary in order to be able to detect the first signs of a further development of the case in the sense of a diffuse organic disease of the central nervous system.

We have thus gained the basis for correctly appreciating the significance of the prodromal stage of paralysis. To repeat, it consists of a series of nervous exhaustion symptoms in the psychic and somatic fields. It coincides clinically with the well-known pictures of neurasthenia and acquires a more fatal significance for us as soon as we have realized that it has arisen on the ground of syphilitic infestation. For these are the cases of luetic infection, from which

in about two thirds of the observations. However, we are not justified in labeling every person suffering from syphilitic neurasthenia as an incipient paralytic, since the disease can persist permanently at this stage of development or even undergo a far-reaching improvement. Indeed, we do not even have the right to pronounce the diagnosis of paralysis if this neurasthenic preliminary stage is accompanied by a single symptom of loss which indicates a circumscribed total damage to one of the infracortical (basal or spinal) reflex centers.

I may well dispense with a more detailed description of this neurasthenic preliminary stage here, as the individual features of the disease will be sufficiently appreciated in the chapter on neurasthenia. I need only point out that you will find all the different varieties of the clinical picture (either pure or in mixed forms) which we know as clinical types of neurasthenia. You will find the neurasthenia with predominantly psychic (affective and intellectual) disorders, the motor, the dyspeptic, the angioneurotic and the sexual form.

I have deliberately spent so long on these seemingly insignificant, uncertain precursor symptoms because, in my personal experience, this is the root of many a medical error and disappointment. Soon the beginner overshoots the mark and diagnoses the paralysis before it is there, soon he fails to recognize the fatal significance of the precursor symptoms and is not equipped to deal with the onset of serious catastrophes.

The significance of these neurasthenic precursors is even more variable and unclear in all those cases in which a syphilitic infestation can be excluded with certainty. In this case, those hereditary forms of neurasthenia which exhibit the strangest combinations of simple neurasthenic and psychopathic disease states are of primary importance. Here, in full manhood, under the influence of psychical damage or alcoholic excesses or as a result of trauma, progressive organic diseases of the central nervous system occasionally occur, which we must clinically classify as paralysis and which shake the theory that this disease is exclusively syphilitic in origin.

And finally, there is a relatively small group of cases of paralysis in which such constitutional weaknesses are absent altogether, in which the patient is said to have been completely healthy until a few months before his illness, to have suffered no syphilitic infestation, but to have lapsed into paralysis under the influence of the other harmful conditions mentioned above. Here any prediction from the precursor symptoms fails.

2. Initial stage.

Dementia paralytica becomes apparent when certain somatic and psychological symptoms of dysfunction have developed, which guarantee the diagnosis with certainty. Anatomically speaking: Dementia paralytica begins when total cortical damage is clinically detectable. However, you do not have to believe that these seemingly

Such a simple statement does even some justice to the difficulty of clinical work in this disease state.

The pathological-anatomical justification of the initial stage is based on the histological findings of the cases that have been submitted for anatomical examination at this early stage. The current state of microscopic technology allows us to detect pathological changes in nerve cells and medullary nerve fibers, blood vessels and neuroglia with a fair degree of certainty. On the other hand, it has still not been possible to visualize many axonal cylindrical processes and their collaterals, as well as the nerve end trees and the entire infinite wealth of the finest nerve fibres with myelin-free myelin sheaths within the cerebral cortex in sectional preparations using the available staining methods. Both the original *Golgi* method and the more recent improvements to it fail completely, especially with our pathological examination objects, in that they only allow individual nerve cells and their larger extensions (axonal branches and dendrites) to emerge. The normal histological studies carried out on mammals by Ramón y Cajal, Sahli, Weigert and Flechsig teach us that the nerve fiber felt of the cortex contains nerve fibers with a myelin-containing myelin sheath, nerve fibers with a myelin-free myelin sheath and free axcylinders. We are therefore by no means justified in drawing a certain conclusion about the extent of fiber loss from the results obtained by staining the myelin-containing myelin sheath parts according to Weigert*'s* or Pal*'s* method. It is much more likely that even in the early stages of paralysis much more conductive substance has perished than we can discover using these methods. It even seems likely to me that the tissue-damaging toxins have the most favorable point of attack on these finest constituents of the conductive nerve substance and cause destruction here before total damage to the nerve cells is recognizable.

You will already have drawn the conclusion from these brief hints that the transitions between compensable nutritional disorders and the final loss of function-bearing nerve substance have not yet been scientifically established and that therefore, in the field of pathological-anatomical research into paralysis, the determination of the first beginnings of this disease must still be counted among the unsolved problems. [2]

Much more significant for the practitioner are the clinical difficulties that stand in the way of timely recognition of the initial stage of progressive paralysis.

Let's start with the mental symptoms. The most prominent symptom that confirms the diagnosis of paralysis is the intellectual deterioration, the slowly and inexorably progressing imbecility. It corresponds to the aforementioned decline of the nervous elements of the cerebral cortex. As simple as the diagnosis of paralytic feeblemindedness is in pronounced cases of illness, it is laborious and misleading.

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[2] I leave the controversial question of whether the initial pathological processes are to be interpreted as inflammatory or degenerative entirely undiscussed, since it is irrelevant to the consideration just outlined; there can be no doubt that the anatomical correlate of the psychic and somatic failure symptoms is to be sought exclusively in the destruction of the function-bearing nerve substance.

It is important to try to distinguish the first signs of intellectual failure symptoms from cerebral exhaustion states with their subjective manifestations (head pressure, inability to work in the intellectual field, affective irritability, insomnia). Above all, we must be careful not to make pathological affective processes the exclusive basis for the diagnosis of incipient paralysis. You will find the same irritable, hypochondriacal mood in both the neurasthenic and the incipient paralytic, which arises from an increased feeling of illness. Feelings of anxiety and attacks of fear or violent outbursts of anger at slight triggers, hyperalgesia against peripheral and internal nervous stimuli are also characteristic of both conditions. It has been stated as a distinguishing feature that the explosive affective outbursts of the neurasthenic tend to pass over into prolonged unhappiness, ill-humor, gloomy, darkened, dogged views of life, etc., while in the incipient paralytic such an after-effect is said to be absent. However, this does not apply to all cases and I do not wish to derive a definite differential diagnosis from it. Much more important to me is the fact that the outbursts of anger, despair and fear of the incipient paralytic quite frequently lack even a remotely satisfactory motive. The disproportion between the triggering cause and the discharge of affect is particularly glaring in those cases in which the patient falls into senseless states of excitement on the most trivial occasions and then not infrequently commits suicide attempts, which in their arrangement and execution reveal the intellectual defect. The case of a high-ranking diplomat who, in a fit of despair, injured himself in a public urinal with a revolver shot at the onset of paralysis is typical.

The extraordinary fluctuation of the emotional state is also suspicious. The neurasthenic, under the influence of cheerful sociability and under the stimulation of alcoholics, will also be able to display hours of heightened joyfulness, even exuberant merriment. In the incipient paralytic, however, everything is much more immediate, less adapted to the circumstances and, above all, less elaborated: hence a sudden, unmotivated change of mood is also easier.

You see, gentlemen, the close points of contact between the two disease states in the field of pathological emotional reaction and have been able to convince yourselves of the finely graded nuances of the mood picture that have to be taken into account in the diagnosis. In my opinion, the decisive factor is to be found in the disturbances in the patient's judgment of his own condition. Whereas the neurasthenic is a keen, critical observer and assessor who registers and analyzes his mental state with the utmost precision, the paralytic's lack of finer self-observation and self-assessment is conspicuous even at this early stage of the disease. Either the paralytic completely ignores the change in his emotional reaction and is surprised, astonished and hurt when reproaches are made to him about his changed and conspicuous behavior, or his judgment is grotesque, altered and distorted, he makes untrue, altered, fanciful representations of the incidents and scenes with which he is charged. In short, his judgment of his inner mental states is damaged. In the same line is the insufficient critical evaluation of his sensory material, especially his organ perceptions. He arrives at the most adventurous hypochondriacal circles of

imagination, which are not infrequently based on psychic

areas open up the scene of pathological processes. The best known are the hypochondriacal complaints of disturbed bowel activity. If such a patient assures you, with the most violent complaints and lamentations about the pain in his body, that he has not had a bowel movement for weeks and that, despite drastic remedies, nothing has been evacuated, you will have to take a closer look at the case to see whether paralysis is not lurking behind the apparently simple neurasthenia or hypochondria.

The most difficult cases to recognize are those in which there is no marked change of affect of the kind described above, and only a peculiar dulling of the emotional reactions, an increased indifference and disinterestedness both in relation to the events of the patient's own personality and to his immediate surroundings, reveal the clinical picture. At the same time the patients perform their daily professional duties apparently quite undisturbed; the clerk goes regularly to his bureau, the officer to his office, the merchant to the stock exchange. Everything goes on as usual until one of the catastrophes to be described later opens the eyes of the uncomprehending environment to the extent of the psychological change. I would like to draw your attention to the patient I introduced to you a fortnight ago. He was a subaltern railroad official who had allegedly fallen ill only 3 days before entering the clinic. The wife reported that the patient had become quieter for about a year; his formerly cheerful, sociable disposition had changed. He initially complained of fatigue and tiredness in his limbs, stomach pains, sluggish stools and insomnia. She was particularly struck by his indifference towards his children, to whom he had previously been tenderly attached. He could come home from work for weeks without taking any notice of them. He would sit at table in silence, "daydreaming", speaking only when asked, and then waking as if from a half-sleep. The wife noticed a significant increase in his appetite, which was at odds with his constant complaints of digestive problems. He refrained from his usual walks, almost always sat in the corner of a sofa, and could hardly be induced to engage in any of his former favorite pastimes, such as playing cards, reading, etc. When he was not on duty and not eating, he slept. She inquired of his colleagues; they confirmed the patient's "drowsiness", but reported that he performed his duties fully as in earlier times. It was only a few weeks before the admission that he became increasingly forgetful. When he went to the bureau, he would get lost in strange streets and would only arrive there late due to detours. He could sit idle at his desk for hours and was often found sleeping. When he was asked if there was anything wrong with him, he was very surprised. He explained that he felt quite well. He rejected a vacation offered to him as unnecessary. A doctor who was called in explained that this was due to nervous weakness (caused by influenza), which would be remedied after a short time off duty. Suddenly the picture changed: the patient became livelier, more excited, told his wife that he was going to the Paris World Exhibition, that he was going to buy a riding horse. He ran to the inns, ordered fine food and wine without having the means to pay for it. The woman made him consult me. The examination revealed: fixed pupils, paresis of the left palatal arch, clumsy, sluggish, nasal speech with distinct stumbling of syllables; left knee phenomena weakened,

Pain sensitivity reduced everywhere, touch sensitivity preserved. The facial expression was indifferent, dull, the left oral facialis slightly paretic. The patient still gives his life history quite correctly, but denies being ill. He gives quite accurate information about his professional activities and about certain jobs he is currently doing. He then reports in an unaffected manner that he will be riding to the exhibition in Paris on horseback. When asked whether he had the means to do so, he replied: "Well, yes, 300 marks." I took the opportunity to introduce the patient to you immediately and use this example to explain the initial symptoms of paralysis.

I drew your attention to the fact that I would of course not dare to discuss the fatal diagnosis in detail in the patient's presence and describe the further course of events if I was not convinced that this entire lecture and the detailed examination of the patient would not leave any mental traces in him. When I asked him at the end of my lecture what I had said, he replied with a smile: "You were talking about a man who is brain-sick."

In the other case sketched earlier, with a longer lasting neurasthenic episode, the onset of the disease had also become apparent through a conspicuous cheerfulness and excitement of the patient, which had increased within a few days to a restless busyness. The patient wanted to throw big parties, go to the World Exhibition in Paris with his wife and relatives, hire extra trains, etc. Here in the clinic he spoke to you in a shamelessly boastful manner about his sexual needs and his performance. Since his wife was not there, he would have to take a girl with whom he would father beautiful children. I drew your attention to the fact that here you can observe the further obvious, moronic increase in certain characteristic symptoms of illness, which are often the very first signs of mental decline. I explained to you that the most highly developed mental processes, which in the individual development represent the conclusion of the mental personality, namely the ethical, aesthetic feelings and conceptual complexes, suffer damage first. The finer understanding of decency and good manners, right and wrong, moral obligation towards oneself and others is lost. If I may say so, the fragrance of personality is stripped away and what remains is the coarse, human machinery with naked, egoistic impulses and aspirations. You will study these symptoms best if you follow the history of the illness backwards in a series of relevant cases. Let me briefly mention a few examples. One of his comrades reported that an officer, who had allegedly only been ill with pronounced paralysis for a few weeks, had only now become aware of some incidents that had already taken place five years previously. The previously scrupulously clean, orderly and dutiful company commander came to duty in untidy clothes and with increasing unpunctuality. Once, during a rest break, he stood in front of his company and urinated.

In another case, at a time when no one had any idea of the outbreak of the disease, the procurator of a large trading house had signed a bill of exchange which he had drawn up for the purchase of a large object of value. When asked about this, he was quite surprised to find a

He was told that he had made a mistake and explained the whole thing as a misunderstanding. It was not until a year later that the paralytic symptoms were fully developed.

In a third case, a hitherto perfectly orderly and morally impeccable merchant made a shameless attack on a young girl who was busy with domestic chores in his family. At the child's cries for help, the patient's wife rushed over and freed her from the attacker's hands. The man smiled at the incident and could not understand that "such a story would be made out of it".

I will confine myself to these examples and only add that increased erotic tendencies also explain why such a large number of incipient paralytics make plans to marry, become engaged and marry. It is certainly one of the most embarrassing experiences of the psychiatrist when these unfortunate death candi- dates come for consultation with their recently wedded wives or brides, usually not of their own accord, but because the careful eye of the companion has noticed certain traits in the behavior of the husband or bridegroom that could arouse concern. If you have no doubt about the diagnosis, you will be obliged to tell the truth. In such cases, I recommend using the expression that it is a serious organic brain disease, which is usually followed by the anxious question of whether it is cerebral softening. It will then be impossible to avoid expressing the fear that this disease will develop and adding the advice that the patient should not continue his professional or business activities. Quite grotesque complications can arise if the incipient paralytic, in accordance with his ethical defect, enters into a marriage with a person of ill repute, e.g. a prostitute. In this case, it is not the worried wives but the man's indignant relatives who bring about the medical decision.

The cardinal symptom of lack of judgment about one's own condition is the source of euphoria, i.e. the subjectively increased sense of well-being with actually reduced mental and physical capacity. However, this lack of judgment is also the origin of those signs of the disease which make the onset of the disease obvious even to the layman today, namely the haphazard self-overestimation ideas which the patient expresses about his personal capacity and which can culminate early on in imbecile grandiose ideas in the case of exuberant fantasy growths.

This occurs especially when affective states of excitement are added, with a cheerful mood, an accelerated flow of ideas and an increased urge to do things. This is the period in which patients make absurd orders and purchases far beyond their means, engage in business speculations which may destroy the existence of the family within a few weeks. How often is the medical expert in a position to give an opinion as to whether this or that purchase falls within the period of illness or not! But negative megalomania (as L. Meyer aptly called it) also springs from the same Quelle. It is the feeble-minded assessment of bodily processes that ultimately leads to a pronounced delusion of smallness, to ideas of the annihilation of the body.

The destruction of individual body parts or organs or the destruction of personality.

In all cases, in the so-called maniacal as well as in the hypochondriacal and melancholic conditions of incipient paralysis, your main task will be to prove the incipient feeblemindedness, which, as the foregoing explanation shows, manifests itself primarily in a weakening of judgment (simple dementia). I emphasize this mainly because the main emphasis is still often placed on the decline in memory functions. My experience, on the other hand, teaches me that in a large number of cases gross memory disturbances may still be absent after the disintegration or at least loosening or weakening of the associations of judgment in their finest, most perfect elaborations has already taken place for some time. If gross memory disturbances are detectable by the loss of whole groups of memory images, they usually relate to events of the recent and recent past, while the memory images prior to the illness may still be quite well preserved.

The first conspicuous manifestation of these memory disorders is the uncertainty of the reproduction of the memory material, especially in relation to place and time. The individual occurrences are apparently correctly reproduced in the narrative, but the chronological sequence of events is very often incorrect and contradictory, without the patient becoming aware of these errors. Only when his attention is drawn to them does he correct his incorrect statements. Hand in hand with this goes a decline in attention; the patients flag after a short time, lose the thread of the conversation, make long pauses, sink into a peculiar brooding, from which they are again disturbed by renewed questions and links. In addition to these disturbances of memory and attention, there is a lack of orientation. Patients find it difficult to find their way in new situations. One of the most common symptoms is that they can no longer find their way around their room in a hotel or hospital and end up in the wrong corridors and rooms. They even get lost in the streets of their home town and find it difficult to find their way back to their apartment.

These phenomena only recede in cases with initial maniacal states of exaltation and are then concealed by an exuberant talkativeness and senseless busyness. Here, however, the incipient mental deterioration becomes obvious through the content of the reproduced imbecile, fantastically polished ideas.

The differential diagnosis of paralysis versus mania is relatively easy if we consider that pure, uncomplicated mania is extremely rare at the age of 30-45 years, i.e. the age period which is primarily associated with paralysis.

In many cases the maniacal excitement will appear pure and genuine; nevertheless, if previous analogous states of excitement or marked cyclic mental disturbances can be excluded by the history of the case, and if alcoholism, which produces similar symptoms, is also absent, it will be almost certain that the maniacal state of excitement belongs to the initial stage of paralysis.

If you investigate more closely, you will find sufficient evidence that the seemingly sudden, acute mental disorder is only a paroxystically occurring further development of the "premonitory" central exhaustion states described by me as precursor symptoms and represents the beginning of paralysis. If the syphilitic infection can be proven, all doubt disappears. I said above on purpose,
"if alcoholism is absent", because I remember a case in which the diagnosis caused great difficulties.

On February 22, 1893, a 40-year-old businessman was admitted to the clinic who had a hereditary burden on his mother's side. The patient developed normally both mentally and physically. In 1882 or 1883 he became syphilitic and was treated with sublimate injections. He has been married for 7 years. His wife aborted in the first year of their marriage and then gave birth to a weak child in 1891, who suffered a red rash a few weeks after birth. The patient is said to have been hard-working, solid and not a habitual drinker. He had been upset since the fall of 1892, and since Christmas 1892 there was a change to a lively, cheerful mood. Numerous drinking excesses. For 3 weeks, increasing excitement, loud, renowned speech; quarrelsomeness, expressions of grandiose ideas. He is the greatest man, he wants to take the school-leaving examination to become a Doctor of Philosophy. He considers himself very rich. He uses crude expressions and finds nothing remarkable in them when asked. He sings ordinary songs, whistles in the house, and at the slightest word goes into violent paroxysms of rage. Sleep is often interrupted, in quiet times he complains of lassitude. Appetite very poor. On admission he was extremely excited. Facial expression cheerful. Very talkative: I can think sharply, can entertain a whole company; I could like all people now; I feel very well; I just have to see other people around me; in 8 days I am going to Leipzig to the fair; tonight we are going out again for a beer. Restless at night, wandering about the parlor, whistling a little song.

From the physical status I emphasize the following: face and conjunctiva normally reddened, arteries not rigid. Wide, long scar in the left inguinal region. Large scar to the left of the frenulum. Right tibial surface uneven. A scar the size of a penny piece 10 cm to the right of the navel. Urine acidic, protein-free. Pupils medium wide, equal; direct light reaction prompt and extensive on both sides; indirect: normal on the right, less extensive on the left. Convergence reaction normal. Forehead, eye and mouth facialis symmetrically innervated. Tongue straight, quietly protruding. Palatal elevation symmetrical. Slight tremor of the spread hands. Knee phenomena slightly increased on both sides. Achilles tendon phenomena not available. Normal hand pressure: dynamometric right 105, 110, left 110, 95. Normal gait. No Romberg. Standing on one leg secure. Leg movements not ataxic, strong. Pain sensitivity in the lower legs decidedly reduced, otherwise normal. Speech articulation intact except for very occasional consonant displacements, writing nothing unusual.

March 23: Out of bed a lot. Walks in the corridor in his shirt. Drinks a lot of water. Wants to go to the wine house. Asks for 10 marks. Wants to buy a summer havelock and a red pas- cham cap with a blue Quaste. The excitement lasts another 3 days.

He then becomes quite calm, orderly, has a relative insight into his illness by admitting to being very agitated.

to have been.

April 27: Achilles tendon phenomena from time to time recently available.

May 1: Conspicuous euphoria.

May 5: Both pupils react somewhat sluggishly.

May 12: When speaking, strong tremolos in the vocals and frequent repetitions of words. Letters home noticeably lacking in content, but reasonably stylized.

May 16: Stronger hesitation when speaking. He confuses names. Memory for recent past decidedly impaired.

An Inunctionscur with Hg was carried out from March 24.

The second patient, who was discharged in the spring of 1893, was admitted to the clinic for the second time on May 22, 1896. He had managed his business well since his discharge and is said to have lived a very orderly life. In the last few weeks he was depressed, quiet and shy of people. For several nights he was restless, sleepless, his agitation increased rapidly and he voluntarily entered the clinic.

Status praesens: pupils wide, equal, roundish; prompt and extensive reaction to light and convergence. Eye movements free. Frown almost symmetrical. Mouth facia- lisinnervation predominantly left. Speech articulation: with difficult words slight heaving, movement of the facial muscles. Knee phenomena of medium intensity, equal on both sides. Achilles tendon phenomena: medium intensity on the right, easily maintained, weak on the left. Ulnar nerve slightly sensitive to pressure on both sides. Pain sensitivity low everywhere. Localization defect rather enlarged. Massive static tremor. In writing: "Konstinopel" instead of "Konstantinopel". But is corrected immediately when reading. Reading comprehension intact. The excitement is decidedly less than at the first recording. He is completely clear and organized, knows the doctors and attendants by name from before, remembers the earlier illness, knows the date, time and circumstances of his admission. Gives prompt information about his business affairs. A few weeks ago at the Leipzig trade fair, he took care of his affairs completely correctly.

On May 24: Stronger excitement. Noticeably cheerful, lively, talkative. Declares himself healthy.

May 25: Sudden change of mood. Anxious. Expiration deepened, accelerated. Vague feeling of anxiety.

June 8: Still very anxious. Sweating profusely. Has wet himself. With light opium treatment increasing calmness.

July 7: Knee phenomena present on both sides, predominantly on the left. Achilles tendon phenomena now the same, of medium intensity. Tremor manuum diminished. Oral-facial innervation almost symmetrical; left side more easily fatigued during teething. Arithmetic problems are solved promptly

July 21: Completely clear insight into illness. Physical status: Nothing abnormal, except slight hesitancy with difficult words.

Over the last few years, the patient's family doctor has reported regularly. The patient has always been completely healthy and able to work. Any nervous or mental disorders have never recurred, as I was told only a few weeks ago.

In the above case there can be no doubt that at the time of clinical treatment the mental symptoms of the disease in all the individual branches, both in terms of development and symptom picture, corresponded completely with the picture of the maniacal exaltation of progressive paralysis. In addition, there was the syphilitic infection that had been overcome earlier, the age of the patient; furthermore, individual innervation disorders, especially of speech and tendon phenomena, could also only be interpreted in terms of paralysis. It was only later, after the second stay in the institution, that I became aware that the patient had always had an explosive, slightly stuttering articulation of speech when speaking rapidly and animatedly. The further course of events did not confirm the initial diagnosis. How should the case be interpreted? It is possible that, despite the strange course of the case, our original diagnosis is nevertheless correct in that it is a case of paralysis accompanied by years of remission, which will later break out again and develop further. The fact that the patient has progressive paralysis is, of course, initially only a vague assumption that will only be confirmed or refuted years later. If we want to hold on to this diagnosis, it is striking that the remissions are so long-lasting and extensive that we have to speak of an almost complete restitutio ad inte- grum. The second possibility is that a functional psychosis in the sense discussed earlier has developed on the basis of the syphilitic infestation, which is clinically documented as a clinical psychosis. The course of the disease is also unusual in that it appears to have come to a standstill after the second cycle. The third possibility, that it was a circular psychosis arising from alcoholism, is not supported by the case history. The patient committed drinking excesses at the time of the excitement, which complicated the clinical picture (tremor manuum, increased speech disorder, ethical damage), but cannot be described as the starting point of the illness.

Such an example teaches you most clearly how careful we have to be with the diagnosis and that even if the symptoms are developed and grouped according to the school, an error cannot be ruled out.

It is also difficult to recognize the condition if it is concealed under the picture of a functional nervous or mental disorder which is accompanied by marked inhibition of all mental processes, drowsiness, disorientation and hallucinatory excitement. If this is accompanied by violent motor discharges, so that a more detailed somatic examination is not possible at a glance, and if at the same time the anamnesis shows that pronounced seizures have been present in recent years or months, confusion may occur. We already emphasize here that a certain form of paralysis (the meningitic-hydrocephalic form) can under certain circumstances be mistaken for epileptic mental disorder. But confusion with alcoholic mental disorders is also possible.

disorders is very obvious at this stage of development, as very analogous conditions (dreamlike confusion, motor excitement and hallucinations) also occur here. In particular, the acute or subacute paralysis with incoherence, disorientation, jactation of the contents of the imagination, tremor, choreiform movements give rise to confusion with the delirious states of chronic alcoholism. The obvious confusion with melancholic and hy- pochondriacal mood abnormalities, which are accompanied by motor and psychic inhibition, has been sufficiently pointed out above. It is needless to point out that in this case the collection of the case history contributes most to the clarification of the case. If Lues can be ruled out with certainty, i.e. if the transitional symptoms described between syphilis of the central nervous system and paralysis are out of the question, the scales are tipped in favor of simple neurasthenia from the outset. Cases in which neurasthenic symptoms can be traced back to childhood or have started during puberty usually speak against the development of paralysis. However, we must never forget that these hereditary-constitutional neurasthenics are not immune to paralysis. On the contrary, heredity, as can be seen from the introductory remarks, must be regarded as an essential factor favoring the development of paralysis. We must always bear in mind that about 40-50% of paralytics have a clear hereditary predisposition.

Other harmful conditions than syphilis, above all chronic alcoholism and morphinism, are certainly also of great importance and complicate the differential diagnosis when they are added to neurasthenia, since in them damage to the intellectual faculties, especially to the higher aesthetic and ethical judgments and emotional reactions, occurs in a form quite similar to paralysis. If certain motor and sensory disturbances caused by the chronic toxic effect are complicated by this, differentiation in the initial stage can become almost impossible. In particular, cases with alcoholic peripheral neuritis (Westphal*'s* sign, tremor, ataxia, speech disorder) are to be included here.

Traumatically induced neuroses and psychoses, which occasionally show symptoms of mental depression, blunting of mental processes, dizziness and tremor, also present differential diagnostic difficulties which are only resolved by the further course of the condition.

Of particular clinical interest are the relatively rare cases in which the paralysis begins under the guise of paranoia; it is then accompanied by systemized delusions (ideas of malice, persecution, grandeur), possibly also by hallucinatory states of excitement. Only the most precise anamnesis, in particular the determination of previous syphilitic illness and the most conscientious physical examination can help here.

This brings us to the somatic signs of paralysis. These are essential to confirm the diagnosis. Some of them can complicate sy- philisneurasthenia, but this does not mean that the diagnosis of the disease is already certain. Before I go into their description, I would like to emphasize as the Quintessence of my experience that the initial stage of paralysis

can only be recognized with certainty and unambiguously when the somatic and mental deficiency symptoms of dementia paralytica are present together. I have already emphasized earlier that in all cases of illness in which you suspect paralysis from the patient's history and mental behaviour, you must carry out the most meticulous physical examination, in particular the most precise nerve status. I emphasized that only a small fraction of the innervation disturbances which you find in this relatively early stage of the disease are already sharply defined and immovably fixed, and that the majority of them are indeterminate and subject to frequent changes in intensity and localization. It is precisely these even less developed signs that require a great deal of training on the part of the physician in order to correctly assess and recognize them. However, it is also necessary that you observe the patient inconspicuously for a longer period of time and draw as little attention as possible to the examination. The best way to do this is to sit opposite the patient, let him tell you his medical history and observe him as closely as possible. You will be able to form a picture of the innervation of his facial muscles, his eye movements, pupil width and especially his verbal expression, which will then be followed by a more detailed examination of the case. In this way you avoid making the patient self-conscious by the examination, and can prevent those inhibitions or increases of motor innervation processes caused by heightened affects, which have already given rise to many deceptions in the examination of neurasthenics and hypochondriacs. In addition, you will gain the advantage that the patient, who has not been informed of the purpose of your examination, will not be forcibly restrained and will not for a short time suppress symptoms of illness which he otherwise presents, e.g. slight tremor, fibrillar twitching, walking and speech disorders, by increased exertion of will. Remember that in all such cases a brief, cursory consultation is not sufficient, that you must inform yourself thoroughly and in repeated examinations and discussions about the mental and physical condition of the patient before you allow yourself a final judgment. In discussing the physical symptoms in more detail, I will take up the examination of the court officer mentioned above. I shall follow the same course that you will best follow in your later medical work. You can contrast one group of cardinal symptoms with another group of more secondary symptoms. The cardinal symptoms are: I. disturbances of pupillary reaction, II. disturbances of mimic facial innervation, speech and voice, and III. disturbances of tendon phenomena.

I. Notice in our patient: The eye movements are free and calm, the right pupil is narrower than the left pupil with a parallel eye position and the same exposure. The light reaction on the left is prompt and extensive, while on the right it is sluggish and not very extensive. The convergence reaction is completely normal on both sides. Let us dwell a little longer on these eye symptoms, as my teaching activity over a number of years has convinced me that the exact observation and correct assessment of these disorders often cause the greatest difficulties for beginners, especially in the initial stage of paralysis. First of all, you must determine whether any eye diseases, in particular iritis or

eye injuries, the condition of one or both pupils has not already been abnormal in the past; whether the pupils have exhibited disturbances with regard to both shape, width and reaction; furthermore, whether the pupillary reaction has undergone an artificial alteration within a short time prior to observation through the effect of drugs (internal or external), e.g. opium, morphine, atropine, eserine. It must always be borne in mind that differences in pupil width also occur in individuals who show no signs of organic nervous disease. [3] Thus you will find congenital asymmetry of the pupil width, especially in connection with pronounced asymmetries of facial skull development or facial nerve innervation, or finally in connection with irregular spotting or uneven coloration of the iris. It may then be concluded that this unevenness of pupil width (with completely normal pupillary reaction) is a sign of disturbed bilaterally symmetrical development (sign of degeneration). You know from clinical experience that these signs of degeneration can only be given a certain aetiological-clinical value if they are found in large numbers in one and the same individual and if the features of a pathological development are also recognizable in the psychological field. The signs of degeneration are of no value in establishing the diagnosis of paralysis. In the various so-called functional nervous disorders (neurasthenia, epilepsy, migraine) you will much more frequently encounter a slight pupillary difference which varies greatly in intensity and is most probably due to functional disorders in the sympathetic nervous system. It is also irrelevant for the diagnosis of paralysis as long as no disturbances of the light reaction are detectable. The same applies to individual fluctuations in the absolute pupil width. Abnormal dilatation of the pupils is very common in anemic, nervous persons and in hysteria. Abnormally constricted pupils (the size of a pinhead) are extremely rare, if we disregard the effects of drugs such as morphine. If they do occur, however, they usually give rise to the suspicion that they are early symptoms of paralysis. In such cases you will have to check the light reaction particularly carefully.

As you can see from these preliminary remarks, the main emphasis must be placed on establishing the light reaction. This arises from the experience that reflective pupillary rigidity is one of the most frequent and most certain early symptoms of progressive paralysis. The same consists in the fact that the pupils do not constrict at all (or only incompletely at the beginning of this disorder) in the presence of light, whereas the constriction is undamaged in the case of accommodation and convergence of the eye axes. However, as today's case shows, this pupillary disorder does not develop all at once, overnight as it were, but rather takes place extremely slowly and, most importantly in this respect, unequally in both eyes. The preliminary stage of reflective pupillary rigidity is pupillary inertia, both in terms of the extent and rapidity of the reflective pupillary reaction. You must also take into account the fact that in the early stages of the disease these disturbances of the pupillary reaction are subject to great fluctuations in intensity, in that one eye or the other may show the symptom of pupillary inertia in different ways.

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[3] The test is of course carried out in medium intensity diffuse daylight and with equal exposure of both eyes.

intensity; at other times, however, a clear judgment in this direction cannot be obtained even with the most careful examination. It should also be noted that both the extent and the promptness of the pupillary reaction also show the greatest individual differences in healthy people. While young, easily excitable (nervous) people show the most extensive and rapid reaction, especially if the absolute pupil width is very large, others, even in the middle years of life, with otherwise completely intact health, have a less extensive pupillary reaction when examined only in diffuse daylight. I advise you to place less emphasis on the extent and more on the rapidity of the pupillary reaction. In advanced years - already beyond the age of 45 - the pupillary reaction begins to become less extensive and also slower. I have observed this primarily in men who are heavy smokers or who regularly consume considerable doses of alcohol without being drinkers in the usual sense of the word. It is therefore not possible to draw a reliable conclusion about paralysis from a slight inertia of the pupillary reaction that is equally present in both eyes. You will now understand why I used the expression "unequivocal judgment" above.

However, if the pupillary reaction is different in both eyes, then the pupillary inertia becomes more important and, if there are other significant suspicious factors for the development of the paralysis, will carry more weight. If one-sided total reflector pupillary rigidity is found, I consider the diagnosis of an organic brain or spinal cord disease to be just as certain as if it is present on both sides. I adhere to the principle stated in my text-book on neurasthenia, that reflective pupillary rigidity does not occur in functional nervous disorders. However, I have already drawn attention there to the difficulties which arise in diagnosis from the combination of past circumscribed organic lesions of the central nervous system with neurasthenia, and have cited cases in which reflective pupillary rigidity was present alongside neurasthenia as a residual of syphilitic processes which had occurred many years ago.

Therefore, this symptom alone cannot guarantee the diagnosis of progressive paralysis with absolute certainty.

This only raises the suspicion of paralysis and, as I will add immediately, of tabes incipiens. Finally, we must not overlook the fact that other organic diseases of the central nervous system, above all multiple sclerosis, and then c e r t a i n focal diseases (tumors, hemorrhages, softening foci) may also show pupillary inertia and rigidity. Likewise intoxications with alcohol, morphine, lead; here, however, it is noteworthy that the eye disorders disappear again when the toxic effect is removed.

Now that we have discussed the diagnostic significance of this symptom in more detail, I would ask you to examine a number of paralytic patients for any pupillary disturbances under my supervision. We are using diffuse daylight on this bright summer day. I have already pointed out the sources of error which must arise from the outset due to unequal exposure of both eyes. When carrying out the eye examination, I would ask you to always use very specific and uniform

Proceed in the same way. In order to rule out convergence and accommodation reactions, have the patient look straight ahead with parallel eye axes. It is best to designate a distant point for fixation. Cover one eye completely with one hand, but without exerting pressure, while you place the little finger of the other hand firmly on the upper orbital rim of the eye to be examined and close the eye against the light source with the palm of your hand. You then lift the upper eyelid slightly with your thumb, without exerting any pressure on the bulb, and observe the pupil width from the inner corner of the eye with the eye in shadow. By turning the hand upwards, it is easy to expose the pupil and allow the light to act for a moment. You then note the light reaction that occurs or does not occur and immediately shade the eye again in order to be able to control the subsequent dilation. Repeat this test several times in short intervals until you have formed a sufficient judgment. On cloudy days, it is essential to repeat the examination in a dark room with an artificial light source using a collecting lens in order to arrive at a definite judgment.

You then immediately follow this examination with a test for accommodation and convergence reaction by holding one fingertip of your hand against the patient and slowly moving it towards the root of the patient's nose. You must also repeat this test several times in order to determine the alternating constriction and dilation of the pupils. The pupillary action test is supplemented by the indirect or consensual light reaction, which occurs when the pupil of the other eye also constricts when one eye is exposed to light. The failure of the indirect light reaction is often the first sign of the onset of reflective pupillary inertia or rigidity.

For reasons of expediency, we combine these exercises, which are carried out in order to test the pupillary reaction, with the examination of the eye movements in order to be able to detect disturbances in the innervation of the external eye muscles at the same time. Among the 5 patients I have ordered to undergo these examinations, you will immediately notice the fourth patient, in whom the left eye shows a clear strabismus convergens. You will see from the anamnesis that the 40-year-old patient contracted a syphilitic infection 10 years ago and suffered an abducens palsy of the left eye 8 years ago. In the following years he is said to have been completely healthy both physically and mentally and to have carried out his professional activities perfectly. For the past six months he has complained of a decline in memory, dizziness, rheumatoid pain in his lower extremities and stumbling in the dark. His wife had been noticing a change in his character for 4 months, in that the formerly evenly calm, orderly, conscientious man showed increased irritability with sudden changes of mood, daydreamed thoughtlessly, slept in company and finally could no longer carry out his professional duties. He frequently lost money, made inappropriate purchases, e.g. he bought a large number of artistically worthless pictures for 4000 M. and children's shoes for 100 M. children's shoes. He voluntarily underwent treatment for his "nerve pain" - he did not notice his mental change himself
- to the clinic. You find absolute pupillary rigidity in the right eye, pronounced pupillary inertia in the left eye and, in addition, the paresis that is still detectable today

of the left abducens. The patient succeeds in bringing the left eye almost to the outer corner when looking to the left; however, n y s t a g m u s - l i k e movements occur. When the eye is at rest, or when trying to fixate on a distant point, the left eye moves towards the inner corner of the eye. Unclear double images occur at a distance of two fingers from each other when the eye moves to the left. I would also like to draw your attention to the fact that these eye muscle tests must not be omitted in cases that appear to be paralysis. However, you must also carry out the visual acuity test, as you will see later that optic atrophy is not uncommon in the group of taboparalytic patients.

II. Disorders of mimic facial innervation, speech and voice. In the initial stage of paralysis, these are motor disorders which can present the greatest diagnostic difficulties due to their incompleteness and their changing localization and intensity. This is because there are no gross, obvious signs of paralysis in the patients, but rather subtle coordination disorders which, at least at this stage of the disease, only rarely (as a consequence of paralytic seizures) lead to damage to simple isolated muscle movements. These can often only be recognized when compound forms of movement are performed, which require a finely graded interaction and a lawful, strictly ordered sequence of the activity of individual muscles, or muscle groups, located in the most diverse innervation areas. This coordination takes place in the motor areas of the cerebral cortex and can be influenced by a wide variety of influences even in healthy people, especially by affective stimuli and by mental and physical fatigue. If these coordination disorders are therefore only pronounced to a lesser degree and of a transient nature, if they affect people who have already been easily excitable in the past and have shown disturbances of motor innervation under the influence of increases in affect, their diagnostic value is low if other paralytic symptoms of disease are absent. The case of Kaufmann, which I sketched earlier, shows you most clearly how misleading this group of symptoms can be. After these preliminary remarks, let us return to the examination of our patient. Let us first look at the innervation of the facial muscles: frowning (in the stimfacialis area) occurs symmetrically. The right nasolabial fold is slightly shallower than the left. Mimic movements and lip movements during speech are predominantly those of the left side of the face. The tongue is stretched out straight, but shows strong rolling and twitching movements. At the tip of the tongue, you will occasionally see finer trembling movements. The soft palate is lifted symmetrically and extensively. If you open and close your mouth in rapid alternation, pull up your upper lip, clench your teeth, sharpen your mouth, stretch your lips forward like a trunk (preferably after pronouncing a prolonged O), you will notice after a few exercises that the finest, fibrillar trembling movements in the right upper lip are inserted into the movements and follow them. If the exercises are continued for 1-2 minutes, the paresis of the right oral facia becomes clearer due to a slight drooping of the corner of the mouth and a lowering of the nasolabial fold. Please note the condition of the teeth during all these tests.

A slight drooping of the corner of the mouth and a flattening of the nasolabial folds is often caused by the absence of one or more upper teeth in one or other half of the mouth. You must also always consider congenital asymmetries of facial nerve innervation if there are imbalances in facial expression when the face is at rest. Here, only the most precise anamnesis will protect you from errors. If, on the other hand, it is a question of acquired pathological innervation processes in the sense of paralytic coordination disorders, this will, as mentioned, only be recognized in the case of finer coordinated movements and confirmed by the occurrence of fibrillar twitches in the paretic muscles. You must pay particular attention to the levator labii sup. alaeque nasi muscle and the quadratus menti muscle. But here, too, you must always ask yourself whether it is not a question of motor discharges of psychological affects in this muscle area, or whether you are not dealing with an alcoholic in whom the mimic innervations can trigger quite analogous trembling and vibrating movements in the area of one or both facial muscles. As unstable, heightened affects are very frequently found at the beginning of paralysis, and alcohol excesses are quite frequently committed by the beginning paralytic, the most varied mixed pictures occur, which make it almost impossible to clearly establish the diagnostic significance of these stimulus phenomena.

Speech disorders are much more serious. For the coordinated movements of the muscles of the larynx, lips, tongue and palate, which are already necessary when pronouncing a single letter (litteral coordination), even more so when connecting the letters to form a syllable (syllabar coordination) and finally when connecting the syllables to form a word (verbal coordination), require such a varied and finely structured interaction of cortical excitations in the motor speech region that even the first beginnings of disturbances of these coordinations are noticeable to the expert examiner. Of course, the principle also applies here: the finer the instrument, the more sensitive it is to damaging influences of all kinds. This explains why even slight deviations from orderly mental activity, e.g. as a result of mood swings or mental fatigue or as a result of various kinds of poison (alcohol, morphine, etc.) can cause noticeable speech disorders. Tone and timbre, as well as literal, syllabic and verbal coordination then show the most diverse disturbances in countless variations. In each individual case, however, we must also take into account the dysphasic and dysarthric symptoms that are summarized as stammering and stuttering (spasmodic and paralytic errors of sound formation, Kussmaul). These occur in neuropathic individuals, especially during childhood and pubertal development, and persist to a greater or lesser extent later on. Only after we have eliminated all sources of error in the clinical examination by taking an accurate history will we be able to fully appreciate the significance of an apparently mild speech disorder for the diagnosis of paralysis. A distinction is made between three groups of pathological manifestations in paralytic speech disorders:

1. the simple littoral coordination disorder when pronouncing consonants, which represent the beginning of words or even just syllables. This gives the speech a halting, hesitant, torn-off quality (hesitation);

2. a kind of atactic aphasia, which manifests itself in the transposition, in the looping and omission, in the repetition of some consonants (brigade instead of brigade, steamship instead of steamship);

3. the same ataxic aphasia in relation to whole syllables, e.g. Dampfffftschepschart instead of Dampfschiffschleppschifffahrt. Taken together, the two latter forms of aphasia cause the clinical manifestation of syllable stumbling. The first onset of paralytic dysphasia occurs during periods of mental and physical fatigue and differs from the analogous disorders that can also be observed in healthy people as a symptom of nausea only in that it occurs more frequently and more intensely. Also, the increase of the disorder due to minor affective excitations is a very important sign of the disease. After some practice, you will become aware of some of these speech defects during longer conversations, even while the patient is telling you his medical history. Even if you have the patient recite a poem or read something from the newspaper, you will be able to recognize these speech errors easily. I attach much greater importance to these forms of observation than to the recitation of very complicated, difficult combinations of words, of which the "third artillery brigade on horseback" is the most common. Also recommended are: "steamship towing" and "infantry reserve exercise". With all these language samples, the patient notices the intentionality and is easily frightened and excited. The affect disorder is then added to the simple paralytic speech disorder. Very often the patient is already aware of the significance of such a speech test and, especially in doubtful cases, e.g. in neurasthenics and hy- pochondriacs, gives new impetus to the idea of being paralytic. The feeling of fear caused by the idea of illness can then cause the most severe hesitation and syllable stumbling and lead the examiner on the wrong track. You will also come across patients who have already been examined in the same way by other doctors and who have already prepared themselves for these exercises. You will then notice that the patients reproduce the key words with conspicuous fluency, while they are shipwrecked with other, often much easier word combinations. So be careful when using these paradigms for paralytic speech disorders and do not overestimate their value.

The disturbances of speech coordination are already intensified in individual patients in the initial stage, in that the above-mentioned fibrillar twitches of individual muscle fibers and more extensive twitches of whole muscle bundles also occur in the articulatory (as in the mimic) impulses and thus the speech, especially the lip sounds, take on a peculiarly tremolo-like character. I have already drawn attention to these disturbances, although they are much more frequently encountered in fully developed paralysis. For there is a group of patients in whom the paretic and irritable symptoms in the motor field may precede all other symptoms of the disease. I remember a patient who came to me a number of years ago with his family doctor to be examined for his nerve condition. The pupillary disturbances, the paresis of the right facialis, the twitching, thrusting movements of the left-sided paretic tongue, but above all the severe hesitation and the pronounced syllable stumbling and letter displacement left me in no doubt even on a cursory examination

In view of the patient's medical history (luetic infection, two paralytic seizures, irritability, slowly progressing paresis), it was a case of paralysis. Before I examined the patient more closely, the college asked me into the next room on some pretext or other, to ask me not to reveal to the patient, either by countenance or words, what I thought of his state of illness; for the patient would observe me with sagacity and deliberation during the further examination, in order to be able to form an opinion of me from my behavior. The college added that the patient was hardly mentally impaired despite his pronounced motor paralysis. Although the patient tended to be more irritable, to have more outbursts of anger and often complained of fatigue during mental work, he still managed his numerous undertakings quite independently, as far as this was still possible given his extremely indistinct speech. Naturally, I complied with the colleague's request. Four weeks later, the patient committed suicide by being crushed by the wheels of a steam engine.

For the sake of completeness, I would also like to draw your attention to the relatively rare changes in the voice as an early symptom of paralysis. There are observations in which a toneless, hoarse, hollow-sounding voice appeared at an early stage. In other cases, a peculiar grumbling speech (aegophony) occurred. In one case mentioned by Westphal, the patient lost his beautiful tenor voice at the very beginning of the disease.

The changes in the handwriting are along the same lines. The family doctor of the above-mentioned patient with the old abducens paralysis presented me with four samples of the patient's handwriting, which contained a request for removal from office as a municipal councillor. Even the heading: "To the chairman of the municipal council" was unsuccessful. He wrote: "To the Lord Chairman", "To the Lord Chairman" and "To the Lord Chairman of the Municipal Council". Here, too, it is predominantly innervation disorders of cortico-motor origin, incomplete and faulty coordination of the writing movements. However, there are also omissions of syllables and words in written expression, which must be related to a decline in mental capacity, especially attention and memory. It is not uncommon to suspect from the letters that patients send to the doctor to register for an examination that they are incipient paralytics. The untidy and unclean manner of letter-writing, numerous ink blots, crossed out and smudged words, ragged, torn, trembling features of unequal size, repetition of words and of final syllables are such typical signs of the disease that one should not fail to have samples of writing from different periods, especially from the recent past, shown in all doubtful cases. Of course, this is only important for educated people who are used to correct written expression. But here, too, one must beware of confusion with alcoholic psychoses, as very similar disorders can be present in the latter.

I would also like to take this opportunity to draw your attention to a peculiar reading disorder, which I have recently discovered to be an early symptom of paralytic reading disorder.

A 48-year-old secondary school teacher, who had been syphilitically infected 20 years ago, fell ill a year ago with neurasthenic symptoms, the most prominent of which were head pressure, severe mental fatigue, poor sleep and dizziness. Various cures brought no lasting improvement. Patient presented himself to me in order to obtain an extension of his vacation. On this occasion his wife - he lives in a childless marriage - told me that in the last few months she had noticed a conspicuous decline in her husband's memory and a high degree of indifference to all external events. He no longer showed any love for her, no longer cared for his friends and relatives, was only ever preoccupied with himself and his illness, and could stare in silence for hours on end. At times he was slightly anxiously depressed and expressed the fear of losing his mind. At other times, he was cheerful, unconcerned and could take great pleasure in the most trivial things. The most striking thing was that the patient, who was used to reading to her in the evenings, was now no longer able to do so: he would start reading, but after only 5 minutes the reading would become incomprehensible by inserting completely nonsensical words which, as she had often been convinced, had no connection whatsoever with the content of the reading. The patient himself had not noticed this until she pointed it out to him. Since then he has avoided reading aloud on the grounds that it tires him too much. On examination, patient complained only of head pressure, inability to think continuously, general physical and mental flabbiness. Pupillary reaction prompt and extensive on both sides, knee phenomenon slightly increased on the left, no facial and tongue paresis, speech slow, tired, toneless, but not paralytically altered. In the psychic field no gross intelligence disorders, no significant memory defects. Patient is completely oriented in time and place, has increased feeling of illness with (quite justified) fear of now suffering from the consequences of his earlier syphilis disease and of becoming incurable.

As you can see, the differential diagnosis between syphilitic neurasthenia and the paralytic disorder is on shaky ground here. With regard to the particular paralytic reading disorder, I am convinced that the diagnosis of progressive paralysis should be made.

III. Disorders of the tendon phenomena. The most significant is the behavior of the knee phenomena. I avoid the more common term patellar tendon reflexes because, as I have already explained elsewhere, this only incompletely characterizes the physiological processes that take place during contraction of the Quadriceps. These are primarily based on the direct mechanical stimulation of the striated muscle fibers. They are also determined by the reflectorically induced state of tension of the Quadriceps. The central mechanisms that mediate and maintain this reflex activity are known to be located in the lower dorsal and upper lumbar medulla. The permanent and complete loss of the knee phenomenon, *Westphal*'s sign, only occurs when the reflex arc itself is destroyed in toto or in individual parts. The permanent loss of the knee phenomenon is therefore one of the most important distinguishing features between functional, i.e. compensable, knee disorders.

and organic lesions of the central nervous system. [4] Temporary cessation of the knee phenomenon can also be observed in functional nervous diseases, e.g. after an epileptic seizure, as a sign of severe nervous exhaustion. If, however, we find the Wesphalian sign in a completely unequivocal manner in one of our patients in whom paralysis is suspected, this is one of the most valuable supports of our diagnosis. However, there are only a limited number of patients in whom the knee phenomena have already completely disappeared in the early stages of paralysis, either unilaterally or bilaterally. First and foremost, there are cases in which the paralysis follows on from an existing tabes (taboparalysis), but also those in which, in addition to the degenerative processes in the cerebral cortex, analogous foci of degeneration have already developed in the dorsal and lumbar medulla at this early stage of the disease. While in the former cases the typical, systematic posterior cord disease underlies the *Westphalian* sign, in the latter there is no mention of such an extension and arrangement of the spinal disease process. We shall see in the anatomical chapter that in the majority of paralytics the spinal cord is involved in the most varied localization, form and extension. form and extent is involved. However, the Westphalian sign will only be present in atypical, asystematic spinal cord affections if the area containing the central reflex mechanism for the knee phenomenon is affected. This is usually only true to a greater extent in the more advanced stages of the paralytic disease process, whereas at the beginning of the disease, even when the lumbar and dorsal medulla are involved, the degeneration processes in the lumbar and dorsal medulla are too small and too irregular in their spatial extent and in their distribution over both halves of the spinal cord to cause a complete bilateral loss of the knee phenomena. Therefore, in the initial stage we much more frequently do not find a complete loss of both knee phenomena, but only an attenuation of the same. The diagnostic interpretation of this finding can be quite difficult. In order to properly appreciate its significance, you must exclude all other causes that can also reduce the knee phenomenon: atrophic conditions of the Quad- riceps musculature, previous injuries to the tendon, neuritic processes, toxic effects

etc. Then you must always bear in mind that the intensity of the knee phenomena is subject to very large individual fluctuations. An organic disease is indicated above all by a one-sided weakening of the knee phenomenon if there are no peripheral causes. If you are able to control a patient with such findings for a longer period of time, you can observe that as the disease progresses, the *Westphalian* sign gradually develops unilaterally from this unilateral weakening of the knee phenomenon and that the other knee phenomenon then also disappears in the same way later on. Of course, this does not often happen in a straight line, but there are numerous fluctuations in the intensity of the knee phenomenon, sometimes temporary improvements, sometimes aggravations. So you can occasionally observe that one knee phenomenon appears weakened, then the other; the final result of the

[4]We have not particularly emphasized here the other causes of *Westphal's* sign, which of course must not be disregarded when assessing the general status praesens (general cachexia, peripheral neuritic processes after infections and intoxications, etc.).

However, the risk of complete loss in the advanced stages of the disease is unlikely to be avoided.

With regard to the technique of testing the knee phenomenon, I would just like to emphasize the following. As soon as you are unable to achieve a moderate knee phenomenon in the patient, you must repeat the test on the bare knee. The patient's foot must be free of footwear to avoid abnormal stress on the lower leg. You must orientate yourself precisely on the position of the tendon by palpation. Your left hand rests on the Quadriceps to detect even the slightest contraction. The patient's attention must be occupied by other things (by t a l k i n g to him about an object of equal importance) and his gaze must be diverted from the knee. You must of course eliminate any arbitrary tension in the Quadriceps muscles. If you have determined the absence or a very considerable attenuation of one or both knee phenomena taking these criteria into account, it is advisable to carry out the examination again using Jendrássik*'s* or Schreiber*'s* aids to control your findings.

Much more common than attenuation or absence is an increase in the knee phenomena in the early stages of the disease. However, the diagnostic value of this symptom is much less certain, as very considerable increases in the knee phenomena are observed in the various functional nerve disorders, especially in neurasthenia and hysteria. Here too, the inequality of both knee phenomena, i.e. a one-sided increase, is much more important for our diagnosis. If it is combined with pronounced spasms in the lower extremity in question and with high-grade dorsal clonus, this finding can almost certainly serve to support the diagnosis of an organic disease of the central nervous system. This finding indicates disease processes in the pyramidal tract located above the lower dorsal and lumbar cord. This is consistent with the anatomical findings, which have quite frequently demonstrated foci of degeneration in the cervical and upper dorsal medulla, in the area of the pyramidal tract, even at an early stage of the disease. Also in cases with combined posterior and lateral cord disease, the increase (unilateral and bilateral) with spastic phenomena of the musculature precedes the loss of the knee phenomena in the initial stage.

The Achilles tendon phenomenon (foot phenomenon) is closest to the knee phenomenon in terms of diagnostic significance. My clinical experience has taught me enough cases in which attenuations and abolitions of the Achilles tendon phenomenon could be observed first, while the attenuation and loss of the knee phenomena occurred significantly later. The examination technique is not easy here either. You have to follow the same rules of caution that I mentioned earlier for the knee phenomenon. Increases in the Achilles tendon phenomenon are subject to similar diagnostic difficulties as those of the knee phenomenon. Here, too, unilateral changes in intensity are more significant than bilateral ones and become particularly significant when there is a pronounced, strong foot clonus.

The anconal tendon phenomenon is far inferior in importance to the previous one. Double-sided increases of the same have no significant diagnostic value. With

unilateral increase, you will always have to think of hemiplegia, especially if the same-sided knee and foot phenomena are also increased.

I hope I have succeeded in shedding sufficient light on the triad of somatic cardinal symptoms occurring in the initial stage. This by no means exhausts the aids to diagnosis; on the contrary, I have the task of informing you of a few more, admittedly inconstant, signs of illness whose diagnostic significance is quite often misjudged and underestimated. This is mainly due to the fact that they only represent a signpost for the diagnosis if they are found in conjunction with one of the cardinal symptoms described above. On their own, they do not provide a strict basis for distinguishing between symptoms of exhaustion and failure with certainty. Even the experienced examiner will only appreciate the full significance of such a symptom, which is often only present sporadically and temporarily at the beginning of the disease, when he looks back at the first beginnings of the disease at a later stage.

This applies above all to the **paralytic attacks**, the significance of which can hardly be doubted at the height of the disease. They also occur in the initial stage of the disease, but then they are almost always of such low intensity and so fleeting that they are sometimes overlooked by the patient and his relatives, and sometimes soon forgotten because of their apparent insignificance. They occur most frequently as vasomotor disturbances, or as migraine attacks, or as abortive vertigo, or finally, in more severe cases, as attacks of unconsciousness, or as mild apoplexy with rapidly passing paralysis. If a syphilitic infection is established by the history in patients affected by these mild attacks, the examiner will, as long as no intelligent defect (in the sense discussed earlier) or none of the 3 cardinal symptoms mentioned is found, just as strongly suspect the development of syphilitic epilepsy (s. str.) or a syphilitic disease of individual cerebral vessels or, finally, circumscribed gummous processes in the soft meninges, rather than paralysis.

Allow me to describe individual features of these paroxystic disorders in a few sentences, insofar as they can be considered larval paralytic seizures of the initial stage. The vasomotor disturbances consist most frequently in sudden congestions to the head with momentary obscuration of consciousness, occasionally also associated with flickering scotoma or with violent ringing in the ears. This is soon followed by burning sensations in the skin of the extremities, vague feelings of anxiety, muscle restlessness, strong palpitations, soon the patients complain of abnormal coldness and tingling sensations in the hands and feet (the extremities then pale and are cool to the touch: peripheral vasospasm); soon the attacks have the character of angina pectoris vasomotoria with radiating pain in the left shoulder girdle and arm. I remember the case of an officer who died of paralysis, in whom such vasomotor disturbances were the only sign of illness for many months before the onset of the disease, in addition to slight mental and physical fatigue. The diagnosis of the attending physician, a

The diagnosis of a recognized neuropathologist was vasomotor neurosis.

These are followed by migraine-like attacks, of which ocular migraine with ciliated scotoma is the most common. However, these are not always the typical attacks (ciliated scotoma with or without lateral hemianopsia, hemicranial conditions, nausea, vomiting). Rather, there are often only simple flickering scotomas with stabbing eye pain or lightning-like, bright zigzag lines (in one or both visual fields) or radially radiating, bright lines with subsequent darkening of the visual field, without being followed by actual migraine pain. Occasionally, however, when studying the anamnesis of paralytic patients, one also finds that pronounced attacks of complicated ocular migraine have opened the disease. I myself know of three typical cases of this kind. One of the patients, an architect, drew the peculiar light figures on the blackboard in the clinic which opened these attacks. In this case, the attacks had preceded the onset of the full illness by six months and had recurred three times.

Severe attacks of ocular migraine are characterized by hemiplegic disturbances of sensitivity and motility, speech disorders and epileptiform convulsions accompanied by dizziness or momentary complete loss of consciousness.

Apart from these larval paralytic seizures of the initial stage, the simple, abortive apoplectic and epileptiform attacks require special mention. They can only be recognized with reasonable certainty as paralytic seizures if there is a brief or prolonged loss of consciousness or a severe dizziness accompanied by a darkening of consciousness and motor deficits. Even if the convulsions or paresis are very fleeting, even if only a few individual muscle groups are affected, the ominous significance of the clinical picture should not be overlooked when the symptoms suddenly appear in patients in their middle years from a state of apparently complete health.

To give an example, a patient came to my consultation with the complaint that he had had several strange coincidences in recent weeks. Suddenly, while writing, he could no longer feel the pen in his hand and had lost control over the movements of the fingers of his right hand; at the same time a feeling of dizziness had appeared. The events lasted only a few moments. Instead of the letters he had just wanted to write, there were irregular, jagged lines. Another form of these coincidences was that the thumb of his right hand suddenly made several twitching movements, which also hindered his writing. A third form of such coincidences was that the patient was suddenly struck by dizziness when climbing stairs, so that he had to hold on to the banister. At the same time, he felt as if his right foot was kicking in the air, even though his foot was placed on a step. It was also impossible for the patient to lift his right foot, as it was paralyzed and "the tip of it was stuck to the floor". It took several minutes for the patient to overcome this weakness. As the man had been syphilitically infected 12 years previously, it was most reasonable to assume that a circumscribed syphilitic infection was the cause.

philitic vascular disease in the motor cortical region had led to these irritation and seizure symptoms. On my instructions, the patient underwent a vigorous lubrication cure, after which the seizures did not recur for six months. Then the patient came to the consultation again, complaining of poor memory and difficulty with mental work, especially arithmetic. At that time, a sluggishness of the pupillary reaction on the right was diagnosed. Six months later, the patient was admitted to the clinic in a violent state of excitement with a blossoming delusion of grandeur. There was now double-sided reflective pupillary rigidity, clearly hesitant speech, right-sided facial paresis and considerable decrease in motor strength of the right hand, clear coordination disorders when writing and with other finer finger movements. After 1 1/2 years the patient died of frequent paralytic seizures.

This example is typical of the majority of abortive paralytic seizures of the initial stage that belong here. As long as no other signs of the disease are clearly demonstrable, it is almost impossible to make a clear distinction between seizures of the same type in the case of specific disease of a brain vessel. The same applies to transient attacks of motor and sensory aphasia and paraphasia, which either occur alone or in conjunction with mild facial and tongue paresis at the onset of the disease. If these apoplectic and epileptiform insults recur several times in the same corticomotor areas, respectively in the muscle areas associated with them, they are almost always followed by a paresis in these areas, which does not consist in a considerable decrease in gross motor power, but rather, as the case just presented proves, only in damage to the finer coordinations. The best way to test this coordination is to perform complicated movements (here too, if the right hand is involved, writing exercises are very useful). writing exercises are very useful) with and without eye closure. The paresis described earlier are predominantly sequelae of these attacks, although the patients are not aware of this connection, as they have usually forgotten the insults because of their insignificance and fleeting duration. But there are also other, no less important accompanying and subsequent symptoms of these paralytic seizures in the initial stage, namely peculiar paresthesias in certain limbs, or sections of limbs, such as tingling, formication, numbness of the arm or individual fingers of the hand, and so on. It is obvious here to think of circumscribed foci of disease in the upper parietal lobe.

Finally, we encounter quite mixed pictures in which abnormal sensations with paroxysms and convulsions are found. They form the transitions to the pronounced paralytic seizures, which occur less frequently in the initial stage, but are not completely absent. Here, too, I would like to give you an example that can replace a more detailed description of the seizures themselves.

In 1892, a 36-year-old businessman was admitted to the clinic who, coming from a family with no hereditary problems, had had an occasional seizure in the third year of his life, but had later undergone completely normal physical and mental development. He served his year as a one-year volunteer. At the age of 24, he married and became the father of a healthy child; another child died[3] year old[4] on the "Ruhr". He was always solid, especially not a potator.

In August 1891 he suffered a peculiar seizure, apparently out of perfect health (he emphatically denies syphilitic infection), without any previous pronounced symptoms of illness. 24 hours after a violent business excitement, while the family was gathered at lunch, he suddenly lost his speech for 10 minutes. He was unable to lift his arm; his hands were also "as if stiff". Since that time, a mental change became noticeable. He was more agitated, more irritable, more absent-minded, and his memory also suffered. He underwent a six-week course of treatment in a mental hospital, which apparently brought him considerable improvement, and in the following months he continued to work undisturbed and, according to his father, completely correct in his business. On January 19, 1892, a second seizure occurred quite suddenly, which his relatives described as a stroke. The patient lost consciousness for a few minutes and was then completely unable to speak or move his right arm for several hours. Both the speechlessness and the signs of paralysis subsided completely within 24 hours. Patient tried to work in business again, but his writing was disorganized, his forgetfulness quite conspicuous. On January 21, he became very agitated, spoke incessantly and incoherently and made violent movements with his arms and legs. The agitation lasted $1\frac{1}{2}$ days. As soon as patient was fit for transportation
he was taken to the clinic.

Status praesens:

January 24. Medium-sized man, $155\frac{1}{2}$ Cm. Earlobes slightly swollen, tongue heavily coated, palatal scar on the left. No glandular swellings. Temporal artery on the right slightly more tortuous and somewhat rigid. Suspicious scarring to the left of the frenulum. Urine protein-free, acidic. Pupils slightly over medium wide, right larger than left. Slight strabismus divergens sinister. Pupillary reactions intact. Nasolabial fold flatter on the left. Left corner of the mouth remains slightly back when speaking and during mimic movements. Tongue deviating to the left in traces. Slight, irregular tremor of the spread hands. No ataxia. Slight increase in knee phenomena and Achilles tendon phenomena. Gait overhanging to the right in traces. The slightest touch is not felt on the trunk and extremities. Marked general reduction in sensitivity to pain. Frequent hesitation during uninhibited speech, only slight hesitation when repeating difficult compound words. No distinct stumbling over syllables. Facial expression cheerful. Informed about personal details, whereabouts, date. Feels perfectly well. "Has a business in Berlin which brings him millions, is Captain of the Reserve," etc. Sleepless at night. Violent states of motor excitement.

February 1st. Anxious today. "They chopped my head off. - I was out of my mind yesterday. - I was in heaven. - God said: "You and your wife are Christian children" and so on.

March 1. Miscalculates 7 times 18.

March 15. Occasionally unclean.

March 23. Does not yet know the name of the keeper of the department.

In April 1892, the patient was transferred to another institution.

If we are to believe the anamnesis, we have here an abortive paralytic seizure as the very first sign of the onset of the organic brain disease, while a second fully developed apoplectiform insult quickly brought the disease to the height of its development. Much more frequently, pronounced epileptiform insults, which are symptomatologically completely similar to genuine epilepsy, precede the onset of the disease. Such insults, if they occur in the prodromal stage several years before the onset of the actual paralytic disease, can be regarded as to a certain extent independent disease processes developing on the basis of "syphilitic dyscrasia". The paralysis then occurs as a further secondary disease of the luetic infection. Such an assumption is appropriate for those cases in which, after the paralysis has become evident, typical epileptic seizures are no longer observed. In other cases, which we attribute to the hydrocephalic-meningitic form, the fully developed epileptiform seizures with profound unconsciousness and with generalized and partial convulsions belong directly to the clinical picture of paralysis and are characteristic features throughout its course.

Special attention should be paid to tabic symptoms (lancinating pain, girdling sensations, circumscribed and diffuse analgesia of the extremities, dissociation of the sensation of touch and pain, ataxic gait, tabic crises, optic atrophy, etc.) in all cases where in the initial stage of paralysis there is a striking disproportion between the somatic and psychic signs of the disease.) in all cases in which, in the initial stage of paralysis, a striking disproportion between the somatic and psychic manifestations of the disease, as well as a strikingly protracted course of development of the condition, interspersed with numerous remissions, justifies the suspicion of taboparalysis.

3. Stage acmes.

The further course of the disease presents no diagnostic difficulties. The signs of the progressive destruction of the cortical elements of the brain may be manifold with regard to the occurrence of the individual psychic and somatic symptoms of the disease, the alternation between symptoms of stimulation, inhibition and failure, the slower or more rapid, the evenly progressive or intermittent course: In the vast majority of cases, doubts about the nature of the condition can hardly arise at the height of the disease. If we first consider the psychological changes described in the previous section, the further manifestation of the mental deterioration consists mainly in the fact that all thought processes increasingly lose their normal connection with the complex of ego concepts. In many cases there is a profound lack, indeed a complete inability, to make associative links between these groups of ideas, which ultimately results in a total lack of judgment about the relationship of one's own personality to the processes of the outside world. The sick speak and act to a certain extent as if in a dream, apparently having no personal relation to their ideas and acts of will, being uninvolved or at least uninterested spectators of their tragic fate. Please note the cheerful or indifferent expression on the faces of

patients who are more advanced in the development of their illness.

During our practical exercise and during my explanatory remarks, I have seen how uncomprehending the patients are, even though I am always forced to point out the significance of the individual symptoms and the overall picture of the disease. In doing so, it was unavoidable to repeatedly draw attention to the most serious significance of the cases. You will often have wondered within yourselves that I unrolled such gloomy pictures in the presence of the sick without taking their feelings into consideration. You were reassured, however, as you could see from the subsequent examination of the patients that they absolutely did not understand the meaning of the lecture, but above all the relationship to their own personality. You remember that the one educated patient reproduced the main points of my lecture with approximate accuracy, the more astonishing it was for you that he could not find the so obvious, almost unavoidable application to his own case of illness. I draw your attention, however, to the fact that this decline in the ability to develop concepts of relationship and to form judgments about one's own personality is usually only fully developed in the higher stages, and that we must therefore be more cautious in our statements about the illness in fresh cases. I therefore had the patients who were still in the initial stage leave before I began my lecture. The disease process just outlined is one of the most important symptoms in cases presenting simple paralytic dementia without affective excitations, without delusions, without hallucinations. A different kind of pathological transformation of the primal associations in relation to the complex of ego representations is shown by both the hypochondriacal-melancholic altered and the maniacally excited patients.

In the higher stage, of course, you will find an even more grotesque manifestation of the most absurd delusions of grandeur or smallness than in the initial stage. The indulgence in millions, the feeling of infinite strength and efficiency on the one hand, the idea of the destruction of individual organs or the whole body and mind, or the idea of impoverishment on the other are typical examples of these disorders. These school pictures of the older authors are, of course, becoming increasingly rare, as I will point out in a moment, and are receding more and more into the background in comparison with simple paralytic dementia. This may be partly due to the fact that simple paralytic dementia was often misjudged in the past and consequently a large number of paralytics, especially in private practice, were classified under other forms of the disease. A further form of mental disorder, which occurs frequently, especially in the more rapidly progressing cases, is the complete dissociation of thought processes. It manifests itself clinically under the picture of rigid absorption with mutism and catatonic phenomena or in violent motor excitations, as well as in jactation of the incoherent content of the imagination and verbigeration.

If we disregard the subacute, stuporous and agitated clinical pictures, we will be able to observe a lawful progression of the weakness of memory and judgment despite the different grouping of symptoms. The above-mentioned characteristic feature of the loss of judgment about one's own personality already indicates the particular nature of the mental decline. The finest and highest concrete and abstract ideas and judgments are lost. The more general a complex of conceptions is, the

more numerous the partial conceptions are.

The more the memory images, of which it is to a certain extent the resultant, must be present, the more conspicuously and prematurely it is doomed to destruction. Furthermore, you will agree that the melting process of the memory images progresses from the periphery to the center, with the most recently acquired ones being lost first. For the beginner it is always most noticeable that such sick people have forgotten the simplest processes, which may have taken place a few hours ago, e.g. visits from relatives, the composition of the midday meal, etc., while those from healthy days have forgotten them. while the memory images from healthy days are often still completely intact. This is of practical importance when you are asked whether a paralytic still has the ability to make a last will and testament. Here you will often hear from the relatives concerned that certain agreements have been made verbally for a long time which, due to negligence, have not been put down in writing. Now that a catastrophe is inevitable in a shorter or longer period of time after the doctor's pronouncement, the mistake made should be made good. Here you have to find out exactly whether you are dealing with the written recording of judgments and decisions of will that originate from the healthy period, or whether the patient is expected to carry out new judgments and acts of will. You will only be able to recognize such a testamentary disposition as fully valid from your medical point of view if you have convinced yourself through detailed conversations with the patient that no new acts of will are expected of him and that the memory images of the will decisions originating from the healthy period are intact. To establish the defect of judgment and memory, it is often useful to make mental calculations with the patient. You will then see that, for example, the multiplication of two-digit numbers fails because the patient is no longer able to retain a calculated number in his memory if he has to continue the mental calculation in order to reach a conclusion. If, for example, 5 x 36 is to be calculated, he forgets the number 150, which he had first found correctly, while he is calculating 5 x 6. The patient may replace this number 150 with a completely meaningless number. It should also be noted that the loss of memory images can vary greatly from patient to patient, with one patient suffering mainly from visual memory images, another from verbal memory images and a third from tactile memory images.

The weakness of judgment is particularly evident when the memory defects are still relatively poorly developed, i.e. when a large number of memory images are still reproduced, but the patient is no longer able to combine them into the simplest judgments. In addition, there is occasionally the occurrence of imaginary visions, which are connected with the memory images in an insane way. Patients can tell whole novels about hunts, dinners, trips they have just been on.

The more acute cases are very often characterized by the occurrence of hallucinatory agitations, sometimes with dreamlike confusion. But even in the slower, chronic cases, isolated hallucinatory excitations are not absent. Grotesque, illusionary reinterpretations of pathological sensations are also combined with hypochondriacal and megalomaniacal ideas. The moral defects characterized in the initial stage become more and more sensual and may lead to the loss of any sense of shame. Furthermore, the emotions of affect are at their height

of the disease and experience a sharper manifestation (outbursts of cheerfulness, anger, depression with moderate anxiety). However, these affective disorders can often be completely absent in simple dementia. There is then a high degree of apathy or a certain moronic contentment from the outset. Characteristic of the patients' imbecility is their increased suggestibility and the mood swings that can be produced to a certain extent experimentally with the suggested ideas. You can make a patient laugh and cry in one breath if you recite cheerful and sad moods in quick succession.

Of somatic disturbances you will soon find a slow, evenly progressive increase in the symptoms of deficits in the motor and sensory area and with regard to reflex processes; soon an intermittent deterioration following isolated or frequent paralytic seizures, which now in their fullest development represent the purest pictures of cortico-motor inhibitory and excitatory discharges. Here you can see very clearly how a circumscribed excitation discharge develops into a spasm of the body musculature, which is sometimes hemiplegic, sometimes generalized, and how the originally clonic cortical spasm is followed by a tonic, also circumscribed or generalized spasm. You see here how the circumscribed cortical spasm begins without unconsciousness, i.e. without diffuse inhibitory discharge of the cerebral cortex, but how the further development of the paralytic seizure is inevitably connected with unconsciousness. You can also see here the peculiar combination of paresis and convulsion, which is also peculiar only to the primary cortical disorder. It is noteworthy that in addition to these primary cortical convulsions, seizures occur which are completely identical to those of genuine epilepsy. In short, paralytic seizures are the best objects of study in human pathology for learning about simple and compound seizure patterns and for paralleling the physio-pathological experiences of animal experiments. Behind these seizures, which are generally referred to as epileptiform seizures, the apoplectiform seizures tend to recede in frequency at the higher stages of the disease. In referring to the earlier description of these seizures, I add here that in many cases it is hardly possible to distinguish them from the apoplectic insults of other organic brain diseases in the seizure itself. We find the same type of development (sudden collapse with complete unconsciousness) and symptoms (sometimes simple hemiplegia, sometimes more complex pictures with hemiplegia, motor and sensory aphasia, etc.) here and there. Only precise research into the history of the case will shed light on the significance of this insult. But the further course of the disease will also soon reveal the facts. Often after hours, usually after a few days, the threatening symptoms disappear; not only the loss of consciousness but also the focal symptoms recede remarkably quickly. Initially, i.e. when these attacks occur sporadically and in long intervals, this regression may be complete. Later on, especially in the case of frequent attacks, the motor and sensory deficits become more and more pronounced after each attack and are associated with more or less pronounced contractures (impaired passive mobility) of the hemiparetic limbs.

A sharp distinction between apoplectic and epileptiform seizures is hardly permissible in the majority of paralytic seizures. Unconsciousness, clonic and tonic

convulsions,

Mono- and hemiplegia, aphasia, etc. appear in manifold and changing combinations.

Quite frequently, series of paralytic seizures are observed, which are characterized by prolonged deep drowsiness, clonic-tonic convulsions recurring at short intervals with subsequent sensory and motor deficits (including paresis of the trunk, pharyngeal, bladder and rectal muscles) and considerable increases in temperature.

Patients often perish in such seizure series, which can last up to 14 days, as swallowing pneumonia and pyelonephritic processes join in. But even with a favorable outcome, recovery from the deep stupor and confusion is gradual and imperfect. If the isolated seizure already causes a clear progression of dementia, this is the case to an even greater extent with these severe protracted attacks. They are not infrequently the signal of an acute onset of final stupefaction.

It is hardly necessary to describe each individual symptom again in its further development. Rather it is sufficient to point out to you that the clinical picture becomes more and more uniform, in that the disturbances of speech and writing become more purely prominent, the pupillary reaction finally ceases, the disturbances of coordination of the ex- tremities become pronounced paresis, and the spinal phenomena (loss of knee and foot phenomena, universal tremor, muscle spasms, dorsal clonus, etc.) complete the clinical picture. Then the whole appearance of the sick person, their facial expression, their posture, their gait is so characteristic that even the beginner is impressed by the clinical picture as a typical one, easily distinguishable from others. That which places the human being at the highest level of development in the phylogenetic series is doomed to destruction here: the upright gait and speech. Please note how the neck often bends and the back curves at an early stage, when the disease has just become more pronounced; the patients appear smaller, slumped forward. This is certainly related to the anatomical change in the motor cortical fields of the trunk and neck region. Add to this the halting, clumsy, often incomprehensible speech and the dead, stupid facial expression, and the idea of Homo sapiens sliding down to a lower level as a result of the destruction of the function-bearing tissue of the forebrain is almost inevitable. The patients lose all understanding of decency, good manners and cleanliness. They eat with unclean greedy haste, stain their clothes with food and, since the bladder and rectal functions have also suffered damage, soil themselves with urine and excrement.

Just a few words about the gait disorder at the higher stage. A distinction is made between a paralytic, a spastic and an ataxic gait. This distinction can only be made clearly in a limited number of cases. A mixed gait disorder is much more common. The paralytic gait is characterized by a general reduction in motor performance. It is unsteady, awkward, swaying, clumsy, shuffling, stumbling. There are also patients who walk with a wide gait or slump at the knees when walking. Especially when making sudden turns, the patient staggers, bumps into chairs, tables and walls, sways forward or backward.

backwards and occasionally falls to the floor. The patient's attempts to climb a chair fail because of the clumsy, awkward, swaying leg and trunk movements. Patients can no longer be allowed to go out on their own, as if there are stones, puddles etc. on the path, they easily fall, partly due to clumsy motor skills, partly due to carelessness, and also through sidewalk crossings, gutters, etc.
w. are easily trapped. These disturbances of posture and gait are pronounced in half of the patients. Especially after repeated paralytic seizures, the predominance of cortico-motor disturbances on one side of the body is very pronounced. Patients are then often completely slumped to one side.

The ataxic disorder occurs predominantly in the earlier stages of the disease and then already indicates a connection with tabic disease (tabic paralysis). In later stages, as the paralysis progresses, this type of tabico-ataxic gait becomes blurred; moreover, it recedes into the background if exaltation states already occur in the initial stage. The gait may then temporarily show a noticeable improvement; however, as the excitement subsides, it changes into the typical paralytic gait disorder.

The spastic gait is most frequently found in patients who have been labeled as trembling paralytics. In these patients, spasms, static and intention tremor, increased knee phenomena, dorsal clonus, etc. indicate the extensive involvement of the pyramidal lateral cords of the spinal cord. However, these groups of motor symptoms can also occur as cerebral (or cerebellar?) symptoms without extensive spinal cord involvement. Mixed spastic-paretic conditions at the height of the disease indicate a combined systemic disease. However, here too we must be careful not to draw strict conclusions from clinical findings to the anatomical diagnosis, as systematic diseases may also be absent. We are still far from being able to draw clear conclusions from the gait disorder as to the type and extent of the paralytic spinal affections.

Sensory and sensitive disturbances take a back seat to motor disturbances, unless they are localized accompanying symptoms of paralytic seizures, hemianopsia, sensory aphasia, etc. The loss of sensitivity to pain and the incongruence between sensitivity to touch and sensitivity to pain, which already appeared in the initial stage, become more and more conspicuous. At the height of the suffering, as soon as their attention can be arrested, the patients can still recognize and localize the finest touches with a fair degree of certainty, while they have become completely insensitive to pain stimuli. It need not be emphasized that this loss of sensitivity to pain is a partial symptom of mental deterioration.

Disorders of general nutrition were observed at an early stage and more precisely determined by weighing the patients. The transitions from the initial to the higher stage are often manifested by a rapid increase in body weight (stage of the embryo point). Even the older authors were aware of the inauspicious significance of this sudden increase in body weight and its causal connection with the progression of the disease.

mental decline. However, the relationship between weight loss and weight gain is not as regular as was previously assumed. Very many cases, especially the agitated forms, never show a stage of embryo point and proceed inexorably towards a progressive decline in strength with increased motor performance and often insufficient food intake. In other cases, especially in cases of severe depression with hypochondriacal delusions and anxiety attacks, a similar continuous decline in nutrition is noticeable. Thirdly, we must consider here cases of simple paralytic dementia without significant motor excitement or change of affect, which always present an inadequate nutritional state even with moderate food intake; these are cases in which severe digestive disturbances and severe visceral pain have already dominated the clinical picture in the initial stage. We believe that the cases in this third category are characterized by extensive involvement of the peripheral, sympathetic and cerebrospinal visceral nerves (degenerative-atrophic processes). They are perhaps closest to cases of visceral syphilis.

Trophic disorders, apart from those of general nutrition, include changes in the epidermoid structures, especially circumscribed hair loss and rapid graying of the hair and beard. This subheading also includes atrophy of the cutis with pigment atrophy and pathological pigment accumulations (nigrities).

Local dystrophies, e.g. mal perforant du pied, and circumscribed muscle atrophies (peroneal area) have been described in taboparalytic cases and in those with peripheral neuritic processes without tabes.

Decubitus ulcers, which are so common among paralytics, have also been described as a trophic disease. A distinction must be made here between decubitus acutus, which may follow paralytic attacks within a few hours and can be observed at the advanced stage of the disease, and the ordinary, I might say, common decubitus, which in former times, when the sanitary facilities of the institutions left much to be desired, almost regularly dominated the pictures of paralysis. In the former, as *Charcot* has also made probable for analogous observations following apoplexy, trophic influences have indeed come into play. In the latter, however, inappropriate care and lack of cleanliness are certainly the main causes.

Whether the conspicuous fragility of the bones and cartilage can explain the frequent occurrence of rib fractures, othmatomas and rhinohematomas in paralytics is still a controversial question. In our opinion, all these cases were undoubtedly preceded by traumatic injuries and it will always have to be investigated whether self-inflicted injuries caused by falling, falling out of bed or rough treatment by waiting staff, etc. were the cause of these incidents. Despite this limitation, we also believe that, as in the case of Tabes, certain pathological changes in the bones and cartilage of paralytics occur in the more advanced stages and cause the increased fragility. This is said to be an increase in the organic components at the expense of the non-organic (neurotrophic basis). The phosphates are said to increase by

40% reduction.

Precise measurements of body temperature reveal in a not inconsiderable number of cases conspicuous fluctuations of subnormal and elevated temperatures, which can be related either to a peripheral disturbance of the thermoregulation centers or to the effects of pathologically altered tissue fluids. Most striking are the sudden, abrupt temperature increases associated with paralytic seizures, which can rise to 40° and above. They are out of all proportion to the severity of the motor stimuli and deficits. The highest increases are found in frequent epileptiform seizures (epileptiform status), which often end fatally. However, even without the onset of paralytic seizures, very extensive increases in temperature are found in paroxystic onset states of excitation with violent motor agitation and absolute incoherence. The most severe collapse temperatures, 32.6-
31.5 measured in the rectum.

Angioneurotic symptoms, which were already noted in the initial stage in the form of vasoparalytic and vasoconstrictor disorders, are still detectable at the height of the disease. Here the vasoparalytic, mostly circumscribed or hemiplegic manifestations predominate, which are characterized by redness, local temperature increase, profuse sweat secretion, in rare cases also by blood sweating in the affected vascular area. General erythematous skin affections have also been observed. The pulse does not show any legal abnormalities, the cardiac activity occasionally shows arrhythmia and allorhythmia.

Secretory disorders include both pathological reductions and increases in saliva secretion. The methodical urine examinations do not yet allow any definite conclusions to be drawn about the metabolic disorders. Intermittent albuminuria, glycosuria and acetonuria are found, as well as propeptonuria. I would just like to draw your attention to the fact that I have recently had the opportunity to observe a whole series of cases in which the onset of paralysis had been preceded by glycosuria for months. These were exclusively cases of pronounced arteriosclerosis. Simple polyuria also occasionally occurs in the initial stage or intermittently at the height of the disease.

Finally, we mention the disorders of sexual function. The increased libido sexualis in the initial stage in the majority of cases, which occasionally represents one of the most prominent early symptoms and can lead to gross sexual excesses as a result of the ethical defects, sometimes increases further at the height of the disease. In other cases, however, a rapid decline in both libido sexualis and potency is observed.

I have recorded the loss of potency quite frequently among the early symptoms of the disease in my notes when it came to cases of taboparalysis.

4. Terminal stage.

To study the final pictures of the paralytic disease process, I ask you to follow me to the infirmary. There you will find a number of seriously ill patients together in the guard room for the unclean and physically paralyzed. The impression you get of them is extremely monotonous. The patients lie dull and motionless in bed. Active, so-called voluntary movements are barely perceptible. The gaze is dull, lackluster, dead, the facial expression meaningless, empty. The attempt to conduct a medical examination with the patients is quickly terminated. Either they no longer turn their heads towards the questioner when called, they appear to be completely unaffected by everything going on around them, or they look uncomprehendingly at the questioner and only answer after repeating the question several times, uttering a few words in a clumsy, awkward, slurred, usually completely incomprehensible language. Requests for arbitrary movements, e.g. to raise the arm or leg, are either not followed at all or only imperfectly. Trembling may also occur when attempting active flexions, extensions and lifts. Passive movements are usually easy to perform. In a group of patients, however, marked spastic tension occurs in the antagonistic muscles of the moving limbs. The tendon phenomena are completely absent in some and extremely increased in others. There is usually absolute sphincter paralysis. Independent feeding is impossible, patients must be fed. Food may only be fed slowly and in small portions, as the patient has difficulty swallowing due to the existing pharyngeal paralysis and there is a risk of food particles entering the larynx. The fatal outcome is not infrequently due to swallowing pneumonia; with careless feeding, but also at a time when the patients can still bring the food to their mouths themselves, there is a danger that large pieces of food will remain on the epiglottis, compress it and thus lead to asphyxiation. It is therefore a long-established practical rule to run one's fingers into the mouth of all paralytics in the terminal stage, when the patient is found in asphyxia and in danger of death, and to search for such food residues. If it is not paralytic seizures that end the patient's life, it is not only swallowing pneumonia but also cystitic and pyelo-nephritic processes following bladder paralysis or infections associated with decubitus. If these dangers are avoided, death occurs through the gradual extinction of vital functions.

Diagnosis.

The difficulties of diagnosis, particularly in the prodromal and initial stages, and those factors which make it possible to distinguish paralysis from the adjacent conditions of neurasthenia and hypochondria, as well as cerebrospinal lues s. str., have been sufficiently characterized in my previous remarks. At this point, I would like to point out that paralysis must be clearly distinguished from postsyphilitic dementia, which develops following the expiry of cerebrospinal lues and may be symptomatologically very similar to paralytic dementia. The essential distinguishing feature here is that

paralysis is an exquisitely progressive condition, whereas postsyphilitic dementia lacks this tendency and persists for years in a completely unchanged manner.

Furthermore, the degenerative processes of the brain that develop on the basis of arteriosclerosis, which are also progressive, present diagnostic difficulties in that they can lead to mental irritation and failure symptoms that are strongly reminiscent of the symptoms of paralysis. Diffuse arteriosclerotic cerebral degeneration and encephalitis subcorticalis chronica are the most common. I must refrain from discussing these forms of disease in more detail, but will content myself with pointing out that the criteria for differential diagnosis lie in the particular form of the clinical picture and not least in the anatomical findings.

In the juvenile forms, the differentiation of diffuse primary cerebral sclerosis is only possible on the basis of the anatomical findings, and even then not always with certainty.

Multiple sclerosis can be confused with paralysis in those cases in which numerous smaller and larger sclerotic foci are located in the cerebrum. It is then difficult to separate the mental and somatic symptoms of irritation and deficits, including the paroxystic symptoms, from those of paralysis. However, there are also cases of paralysis that can clinically mimic the symptoms of multiple sclerosis. The section then only reveals the typical changes of paralysis.

Precise examination and especially research into the development of the disease protect against confusion of paralysis with focal diseases of the brain: brain tumors, thrombotic, embolic softening, hemorrhages, abscesses, etc..

Course.

In the majority of cases, the disease can be described as exquisitely chronic, leading to a fatal outcome in a variety of ways. Whether there are any paralysis cases at all that come to a definitive standstill or to a relative cure with a greater or lesser defect is still a controversial question. In my experience, there is only one, the so-called taboparalytic form, which even shows years of arrest at a relatively early stage of cerebral involvement in the disease process. There are also isolated cases in which certain somatic symptoms of paralysis are associated with acute states of mental excitement, size delirium, disorientation and motor excitement. After the excitement stage has subsided, complete mental clearing occurs. The somatic failure symptoms, loss of knee phenomena, reflective pupillary rigidity, persist without the presence of pronounced tabes. In the mental sphere an apparently complete restitutio ad integrum is achieved. However, the attentive observer notices that a decrease in intellectual capacity, albeit to a lesser degree, has remained, which is noticeable by rapid fatigability during mental and physical exertion and a series of persistent neurasthenic complaints. In such observations, previous syphilitic infections can always be

can be determined. I myself have only observed one case belonging to this category, which could be included in the group of cured paralytics with this restriction. It concerns a senior teacher who has now been active again for 15 years in a literary and practical capacity, albeit without working at a public school. In a classroom he cannot teach for one hour at a time, allegedly because of the bad air, without experiencing head pressure and dizziness with flim- merscotoma. The patient still has *Westphal's* sign, other tabic symptoms are absent. It can only be explained here that the tissue damage caused by the syphilitic disease was limited to a few foci in the central nervous system after the acute attack, which was often caused by toxic effects or vasomotor disorders, had subsided. However, it will not be possible to give a definitive verdict on the case outlined here, as there is of course still a risk of the condition recurring. In any case, in the majority of similar cases which have been listed in the literature as cured, the disease has subsequently flared up again.

The average duration of progressive paralysis has been calculated at $2\frac{1}{2}$ - 3 years. In individual cases, it varies between a few months and two decades.

As far as the age of the patient is concerned, paralysis occurs less frequently as an early form (juvenile form) on the basis of hereditary Lues. It is usually a disease of the period of life in which the individual has reached maturity and is at the height of his capacity. There is no sharp boundary against senile involutional disease. Transitional forms between the simple presenile and senile dementia states and the arteriosclerotic brain degenerations also occur here.

The acute, so-called galloping and subacute cases, which are characterized by a stormy course, form a separate group. In addition to the profound dissociation of the psychic processes with hallucinations and illusions, they are characterized above all by violent symptoms of irritation, inhibition and failure. However, there are no new phenomena that are foreign to the chronic clinical pictures, rather there is only an accumulation and compression of the known, previously mentioned symptoms. From a pathogenetic point of view, they always suggest that, in addition to the destructive processes in the function-bearing substance and in the supporting tissue of the central nervous system, general toxic effects, either caused by syphilis toxins or by toxic decay products of the central nervous system, are responsible for the stormy, lethal course.

The chronic cases present such a rich variety of psychic and somatic symptoms in varying groupings and successions that it is not possible to draw conclusions from the particularly prominent symptoms of the disease as to certain directions of progression and certain clinical forms of paralysis. The original, schematic classification of typical paralysis into the three stages of megalomania, raana- cal excitement and terminal insanity has not withstood the progress of knowledge. Due to the special nature of the paralytic disease, it is impossible to draw conclusions from individual psychic and

The division of somatic symptoms into clearly delineated stages can be derived from this. The division we have made into four stages, between which there are no sharp boundaries in practice, therefore has a prevalent didactic value.

However, within the framework of paralysis, taking into account the overall course and the grouping of the symptoms, it is possible to distinguish not only between adjacent conditions, but also between certain interrelated subdivisions of the disease. Of the subforms in question here, the following can be separated from each other with regard to both the course of the disease and the anatomical findings:

A. The meningitic-hydrocephalic form.

Clinically and symptomatologically, it is characterized by the typical, remittent course, by the most severe paralytic attacks with general and localized motor stimulation and paralysis symptoms and by violent states of excitation with total drowsiness, incoherence and disorientation. In striking contrast to the paroxystic phenomena, the initial stage is characterized by extensive involuntary remissions, which often last for months and, to the layman at least, appear to end in recovery. The clinical pictures of idiopathic epilepsy with post-epileptic twilight and excitement states can be feigned. The disease either ends fatally at the height of the suffering following a violent paralytic seizure, or progresses very slowly until complete exhaustion of strength. This variety of paralysis is also similar to epilepsy in that a severe epileptiform seizure can be followed early on by a considerable memory defect.

The autopsy reveals a pronounced diffuse, calloused, swollen leptomeningitis if death has occurred in the final stage. However, even if the disease is terminated early, there are signs of expired diffuse arachnitis with abundant accumulation of serous fluid in the subarachnoid spaces. The cerebral cortex is extremely atrophic. The medulla is also significantly reduced; the weight of the brain is reduced by 200 grams and more. In the final s t a g e s , the ventricles are extraordinarily dilated and filled with serous fluid. Microscopically, the enormous sub- and infracortical fiber atrophy is striking. In early lethal outcome, the degenerative changes in the nerve cells decrease in extent.

B. The hemorrhagic form.

It comprises the majority of subacute cases of paralysis and then has a short prodromal stage in at least some of the cases. In other cases, however, the clinical picture is initially that of simple paralytic dementia; the deviation from the usual course only occurs at the height of the disease. In this subtype, the paralytic seizures recede into the background.

Acute disease states with a high degree of dissociation of the mental antecedents

The most intense and violent hallucinatory excitations are characterized by the particular type of motor stimulation. Sometimes it is a general coarse shaking tremor, sometimes it is pronounced choreatic twitching which accompanies the psychic excitement. In several cases I have observed, these accompanying symptoms have lasted until the end. In another case of this type, which is still in the clinic, the excitement stage has subsided after 7 weeks and the above-mentioned simple picture of terminal insanity now exists. At the level of the disease attacks occurring in the hemorrhagic subtype, there are similarities with the toxic-infectious cerebral diseases.

In addition to the known diffuse macroscopically and microscopically detectable changes in the brain and spinal cord, macroscopically visible larger and smaller hemorrhages that have occurred in the brain substance as well as in the epicerebral and subarachnoid spaces can be seen. Microscopically, there are also numerous miliary hemorrhages within the cerebral cortex, the medulla and the brain stem, sometimes of fresh origin, sometimes only in the form of amorphous or crystalline blood pigments, sometimes in the form of small yellow pigment granules that can be regarded as residues of older hemorrhages. I note in this connection that isolated small remnants of blood are found in the cerebral lymphatic spaces in almost all paralytics who have died in the initial stage.

The special feature of this hemorrhagic form is the early occurrence of massive miliary and larger hemorrhages. The cause of these hemorrhages is an extensive hyaline degeneration of the capillaries, arterioles and small venous blood vessels. It is noteworthy that in one case belonging to this group there was still florid syphilis at the time of the paralytic disease and that in a second case extensive tuberculosis was found.

C. Taboparalysis.

Clinically and symptomatologically, only those cases belong here in which the paralytic disease has joined an already existing tabes. They are characterized by an exquisitely chronic course of the paralytic disease, which is the result of long periods of inactivity. The slow progression of the intelligence defect is particularly striking. Here a quite unusual division of the intellectual damage becomes apparent. For years, only the finer ethical and aesthetic ideas and sensations can suffer a very extensive loss, while marked judgmental disturbances and memory defects are only occasionally detectable.

The tendency of patients to excesses in venere, to lying and cheating, to crude, violent excesses is often the most obvious sign of the change of character. Thinking and speaking, on the other hand, is generally logically correct and often associated with an almost astute reasoning. Patients often know how to cover up their moral defects in the most skillful way. If they are patients of a higher social standing, they can continue their professional activities undisturbed during the remission of the illness.

In the prodromal stage, taboparalysis shows the typical findings of tabes; here, too, reflective pupillary rigidity, *Westphal's* sign, optic atrophy, lancinating pain, gastric and laryngeal crises are the order of the day. It is unnecessary to debate the question to what extent these findings are to be attributed to tabes or paralysis. For we are not justified in making the diagnosis of taboparalysis until the other characteristic features of paralysis have developed in the psychic field and other disorders belonging to paralysis, especially paralytic attacks or intercurrent hallucinatory excitement states, often lasting for weeks, have occurred.

As the disease progresses, the typical generalization of the intelligence defect and the characteristic disorders of writing and speech are also found. You have seen such a case in the clinic, in which the paralysis demonstrably began 15 years ago, while lancinating pain, girdling sensations and a slight walking disorder had already existed for years before the onset of the paralysis.

Anatomically, apart from the tabular changes in the spinal cord, we find an extensive atrophy of the fibers which, as far as my previous experience allows me to conclude, is much more pronounced in the cerebellum and in the posterior cortical sections located beyond the central convolutions than in the other cases of paralysis.

D. The peripheral-neuritic, visceral form.

I would like to use this name to refer to a fourth variety of paralysis, which I will list with all due reserve. It is characterized by severe, general and local nutritional disturbances and by a sometimes more rapid, sometimes slower loss of strength, which is associated with the most severe visceral neuralgia, paresthesia, burning and caustic sensations in the abdominal skin, the back and the lower extremities. In contrast to the taboparalytic form, the knee phenomena are still unimpaired or even increased at the beginning of the disease, but are gradually lost as the condition progresses. In the psychological field, there is a simple paralytic dementia. Patients often make hypochondriacal complaints relating to painful sensations. Here, too, a distinction can be made between more rapidly progressing cases, which end within a year of the onset of the disease, and chronic, protracted cases. At least several cases belonging to t h i s group indicate the possibility of such a distinction. I have presented you with a case in which the visceral form of paralysis has been present for 6 years. Although the patient's dementia is well advanced, the whimpering and complaining about his painful sensations still occur intermittently. At other times, the patient lies dull and motionless in bed. His body weight has steadily increased. At present he hardly differs from the cases of ordinary paralytic dementia. You remember from the history of the case that the development of the disease has been a very peculiar one.

The illness began with severe anxiety, feelings of oppression in the chest and dizziness. This was followed by temporary double se-

and flickering scotomas. In the fall of 1895, memory weakness became apparent. Patient, who was an accountant in a store, often prescribed himself and had to add repeatedly until he got the right result. This was followed by insomnia, heaviness in the legs from the knee downwards, burning, itching skin sensations in the lower thighs, weakness of the bladder, severe pain in the abdomen, radiating into the urethra. The knee phenomena were increased on admission, as were the foot phenomena. Reduced sensitivity to pain in the lower extremities. Numerous pain pressure points on the spine, at the facial nerve exits and in the course of the ischiadicus, on both sides. Speech articulation still intact. Both pupils very constricted. Light reaction clearly weakened on both sides. In the course of the observation the light reaction completely disappeared on both sides. The knee phenomena also disappeared. Patient became more and more apathetic, weaker in memory, complained a lot about pain and fear. He would probably be a criminal and would have to go to prison for indecent acts. Also paresthesia in the soles of his feet, pain in the rectum and abdomen, loss of appetite. Often persistent constipation, which occasionally led to fecal congestion with chills and fever. Frequent complaints of headaches.

The anatomical studies on the cases belonging to this group have not yet led to a clear result. We will have to content ourselves with characterizing these cases anatomically as analogues of peripheral neurotabes. Clinically, the present visceral form would be best characterized as paresthetic and neuralgic paralysis.

Far be it from me to assume that the listing of the four subtypes precisely delineates the extended area of progressive paralysis in all directions. Rather, enough cases remain which take an atypical course and do not fit exactly into one of the four anatomical subdivisions, but which, despite their clinically atypical course, show the pathological-anatomical findings of ordinary paralysis.

Finally, it should be mentioned that the disease usually only manifests itself differently in the initial and climax stages. The final symptoms are usually the same and the anatomical features are often blurred in the final stage. [5]

Pathological anatomy.

The quickest way to illustrate the anatomical findings is to show you the microscopic findings on a series of specimens.

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[5]The attempt to establish a special symptomatologically structured subtype of female paralysis does not seem justified to me, since in my experience female paralysis, apart from the occasional protracted course, does not present a peculiar course. It has also been claimed that female paralysis is proportionally less often caused by Lues than male paralysis. According to my observations, this assertion is by no means correct. It is only to be admitted that paralysis occurs less frequently in females than in males, regardless of the cause. According to current statistics, there are 2.5 to 3 male cases for every female case. In earlier years, investigations had revealed different ratios (1 female: 9 males, respective 1 male: 8 males).

lead. Let me, however, make a few preliminary remarks about macroscopic findings characteristic of paralysis. In cases in the final stage of the disease, the characteristic findings are as follows: Extensive atrophy of the brain, which can be detected on weighing by a reduction in the total brain weight to 1000 grams and less. The cerebral convolutions are narrowed and protrude like a ridge between the gaping sulci. Their surface can have an irregular, uneven, in places bumpy appearance due to the fact that the atrophy is not evenly distributed. The narrowing is most clearly recognizable in the lateral, medial and basal sections of the stimulus brain, whereas it is barely noticeable on the cortex of the occipital lobe in common paralysis. The narrowing of the cortex is even more apparent on cross-sections. These show a grayish-white coloration of the cut surfaces instead of the gray-reddish one. The medullary layer is also decidedly narrower, very often edematously swollen, whitish shiny, of doughy consistency. The ventricles are strongly dilated (hydrocephalus internus), their ependyma finely granulated. The latter is particularly prominent in the fourth ventricle, where the surface in the region of the alae cinereae appears rough and like a grater. However, in the region of the striae corneae, in the vicinity of the foramen monroi and in the infundibulum, ependymitis granularis is only exceptionally absent in old cases. The inner capsule is often discolored in streaks of gray on the sections, the same type of discoloration is seen in the cerebral pedicles. The soft meninges are thickened in the majority of cases, but by no means constantly, sometimes in the form of callous, plate-like deposits in the arachnoid, sometimes as white strands along the vessels. Granular thickenings of the arachnoid are rarer. The subarachnoid spaces contain a large amount of serous fluid. In some cases a pronounced pachymeningitis haemorrhagica is present. When the soft meninges are removed from the surface of the brain, the vessels radiating from the uppermost glial sheath into the pia can adhere so firmly that they are partially torn away. In a number of cases, flat, rust-colored deposits interspersed with fresh blood spots are spread irregularly over the basal cranial fossa and the convexity, but massive accumulations of blood and hematomas of the dura mater are also present.

Macroscopically, the cerebellum shows no clear changes, but degeneration processes in the spinal cord are hardly ever missed. These are often irregularly recognizable on the individual spinal cord cross-sections as streaky or extensive grey degenerations, predominantly in the posterior cords and in the pyramidal lateral cords. The spinal roots also show gray striations. In addition, there are pronounced simple (typical posterior cord or pyramidal lateral cord degenerations) and combined systemic diseases.

In fresh cases, we have to distinguish between: a) acute and subacute, b) chronic cases in which the disease process has been fatal at an earlier stage of the disease (when the disease has demonstrably lasted up to one year) as a result of intercurrent diseases. In acute or subacute cases, the atrophy is usually macroscopically recognizable, albeit to a lesser extent and often concealed by edematous swelling or heavy blood filling of both the cortex and the medullary substance. In the chronic cases referred to under b), any clear change can often be

missed macroscopically. Only the episodes

dymitis granularis has rarely been absent.

Let us now turn to the microscopic changes, which I am convinced are much more conclusive. Here you have to differentiate between the following on the specimens: a) the changes in the function-bearing nerve tissue, b) the glial substance s. str. (ectodermal parts of the supporting substance), c) the blood vessels and the sap tract system.

a) The changes in the nerve substance probably affect the entire neuron. In my opinion, which is of course based more on considerations than on evidence, both the axonal processes and their collaterals (medullary fibers and free axonal cylinders) as well as the dendrites are involved in the degeneration process to a large extent at an early stage. As is well known, we do not yet have any methods to visualize these functionally so important parts of the neuron, on whose integrity the intracortical or intercellular transmission of excitation is based, in sectional preparations. It is even less successful in clearly demonstrating their pathological changes. [6]

On the other hand, we are able to detect the changes in the nerve cells and nerve fibers containing the medulla with sufficient clarity and certainty. I would like to draw your attention to the great diversity of cell degeneration.

You can even find intact cells in old, terminal cases. The larger, pyramid-shaped ganglion cells are most suitable for such cell studies. In addition, cells with partial and total loss of *Nissl's* granules can be found, as well as nuclear alterations with bloating, Quelling and complete dissolution. The nuclei are then fragmented or completely lost. Finally, shrunken, sclerosed cells without continuation are also found. I have already drawn your attention to the significance of partial and total cell degeneration. The loss of the medullary nerve fibers can best be seen in the so-called tangential fibers (uppermost cortical layer). However, it is also unmistakable in the other cortical layers, especially in the deeper radial layer, and also in the medullary ridge.

b) The changes in the glial substance, particularly in the old cases, consist of a conspicuous increase and thickening of the glial fibers, especially in the upper cortical layers. The glial cells are also markedly increased in the outer glial sheath. This also applies to the early stages of chronic cases. In the more advanced stages, the proliferation of glial cells becomes increasingly evident. In acute cases, glial cell proliferation is ensured by the new Weigert method. When chronic cases in later stages of the disease were examined, karyokinetic processes were also observed in the glial cells, which led to the long-known images of spider cells. Examination of the glia has shown that the deeper parts of the brain, the medulla oblongata, the medulla oblongata, the semioval center and the optic thalamus, are also extensively involved in the disease process.

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[6] The *Golgi* method and the more recent improvements to it cannot yet be used to study paralytic brains.

c) The changes in the blood and lymphatic vessels are of great importance and are already present in the early stages of chronic paralysis, but especially in acute and subacute cases. The venous bloodstream is dilated everywhere and bulging with red blood cells. The arterioles and capillaries show extensive regressive changes of the character of hyaline degeneration. The endothelial valves (especially of the small vessels) are thickened; the other parts of the vascular wall also show clear proliferative processes, streaky thickening and nuclear proliferation, especially in older cases. Red and white blood cells, clotted lymph, amorphous, cloddy and fine blood pigment are found in varying amounts in both the extra- and intraadventitial sap tract system, which, especially in older cases, is sometimes greatly expanded, sometimes narrowed in places by moderate hyaline deposits. The study of leptomeningitic changes is also very instructive. One of the first changes here is the thickening and proliferation of the endothelial cells on the vessels radiating from the pia into the cortex. I have also shown you specimens in which you can clearly see nuclear proliferation within the endothelial membrane of the pia itself. In other places you can see partial obliteration of the epicerebral spaces due to adhesions of the glial sheath with the thickened pia section. It is easy to understand that these obstructions of the drainage openings of the extravascular or intercellular juice canal system cause severe disorders of lymph circulation (lymph congestion).

As far as the significance of the anatomical changes is concerned, we may initially assume that the destruction of the function-bearing tissue (in the form of neuronal damage) is the essential and decisive factor for the loss of function. On the other hand, it is doubtful whether the pathological processes on the function-bearing nerve substance or on the glia or finally on the blood vessels are the primary ones. I will avoid going into these controversial points here and would just like to express my conviction, based on extensive studies, that the damage to the nerve tissue is the primary one and that the reparative and exudative inflammatory processes only follow this damage later.

Therapy.

Unfortunately, the most important chapter with regard to our medical tasks is the most meagre in this disease. Since the beginnings of the disease are still often shrouded in darkness, it is not possible to initiate extensive and successful prophylactic treatment with any certain prospect of success. We can only set out general principles. If a previous syphilitic infestation has occurred, if signs of syphilitic neurasthenia or hypochondria have developed, we will have to advise the patient to take the greatest physical and mental care. If there are signs of specific syphilitic disease processes in the area of the central nervous system, we will carry out antisyphilitic treatment, as it is well known t h a t paralysis often accompanies or follows syphilis.

We will subject the illnesses in question to constant control and make even the slightest psychological changes the starting point for stricter medical measures. Above all, we will make the mental and physical diet

tetics.

Once the paralytic disease has been diagnosed, we must first ensure that the patient is carefully cared for. We must then protect the patients and their relatives from material ruin as a result of damage to their intelligence, delusions and mental states of exaltation. We will also ensure that any offences against morality or other criminal acts are recognized in their causal relationship to the brain disease and that the patients are protected from further excesses. Early incapacitation of the patient is necessary to prevent the civil and criminal consequences of the patient's manifestations of illness.

Simple paralytic dementia can very well be cared for in the family if the social conditions are favorable. Excited patients or those with suicidal tendencies require institutional treatment.

I can assure you that the care of paralytics at the height of the disease and in the terminal stage makes the greatest demands on the attention and patience of the doctors and nursing staff.