

*Paralysis -*

GENERAL PARESIS,

OR

INCOMPLETE PROGRESSIVE PARALYSIS.

BY

JOHN P. GRAY, M. D., OF UTICA.



lc

ALBANY:

VAN BENTHUYSEN'S STEAM PRINTING HOUSE.

1866.

HMD  
ALPHA  
BOX  
C.2

Gift  
F. W. Putnam

01

# GENERAL PARESIS,

OR

## INCOMPLETE PROGRESSIVE PARALYSIS.

---

I propose under this head to treat of a disease of comparatively modern origin, at least so far as its recognition is concerned by the medical profession, as a distinct form of cerebral disease. Though it has been recognized under various names for some years, it has not been mentioned in works on general practice, and has received little if any attention in the lectures of our medical colleges. I shall not here enter into the question of its remote or recent origin, or attempt an exhaustive essay, but present, as succinctly as possible, a brief outline of its recorded history, its etiology, sesmiology and morbid anatomical appearances, desiring rather to direct the attention of the general profession to the study of the disease, its grave character, and to the fact of its increasing prevalence in this country. I am induced to present the subject before this medical body from two considerations: first, that of the entire number of cases placed in the asylum at Utica, for treatment, since my connection with that institution, but four cases have been recognized by the medical men in attendance before admission to the asylum; and secondly, that most of the cases have been brought to our care by friends in the confident expectation of speedy recovery, generally based on the opinion of the family physician. These facts would argue that few physicians are familiar with the disease, and that, assuming from the presence of certain psychic manifestations, the existence of mania, melancholia or dementia, they prognosticate accordingly; for any physician having once recognized a disease so marked in its somatic and psychical symptoms, and familiar with its character and progress, would give a positive prognosis of its fatal issue.

General paresis is most frequently designated under the name "General paralysis of the insane." Dr. Saloman, a Swedish physician, has substituted the term paresis, as indicating the paresifying nature of the disease.

By universal testimony, paresis is almost entirely confined to men. Of 119 cases which I have observed, but 8 were women.

It seems to be more prevalent in some localities than others. Dr. Skae, of Scotland, states that in the Edinburgh asylum, under his care, there are generally to be found from 20 to 25 cases, while at Montrose asylum, where there is an average of more than 300 patients, there are none. As the medical head of the Montrose asylum was an assistant under Dr. Skae for many years, he would recognize the disease. This fact has been observed also in America. In the east and north there are more cases than in the west and south.

The distinct recognition of the disease does not date back over 60 or 65 years, though some maintain that they have found symptoms in the writings of several authors and dating back to Willis, in 1672. Esquirol, in 1805,

describes it, and credits Haslam with having first called attention to it. Since that period it has received much attention by writers on insanity, and especially in France. Some authors, among whom are Esquirol, Delaye, Calmeil, Georget, Leubuscher and Leidersdorf, consider that the paretic or somatic symptoms are engrafted on various forms of insanity; merely a peculiar form of paralysis consequent upon and associated with the progress of mental disease. Bayle, Parchappe, Jules Fabret, and more recently Saloman, maintain that the disease is a distinct species—a form of insanity—and in this view I think English and American alienists will agree. Dr. Van der Kolk, one of the latest and ablest among German authors, does not recognize general paresis as a disease *per se*, under this or the more generally accepted name, general paralysis of the insane, but he gives a most faithful description of it under the head of idiopathic mania; so that there can be no doubt of its existence there, and indeed he proves its great prevalence in the fact of having so many cases as to induce him to group them in one great general division. Some authors have maintained that paresis, or paralytic insanity, might exist without the manifestation of paralysis, and under the term “ambitious mania,” have given some cases. “Ambitious mania” is a very different disease, and only resembles paresis in the character of the delusions. If necessary, I could present a number of cases to show this. In a discussion of paresis, recently, in the Psychological Society of Paris, this point was brought up. I quote the remarks of M. Baillarger:

“Esquirol, as M. Parchappe still does, regarded all cases of ambitious mania as simple insanity, as long as they were uncomplicated with symptoms of paralysis. Bayle, on the contrary, and with him M. Jules Fabret, considers many of these cases as presenting a special form of insanity, even before the appearance of paralysis. A case of ambitious mania is reported in the thesis of M. Fabret, which was cured after two years duration, without the patient having presented any evidence of paralysis. But, according to the author, this patient was not the less attacked with paralytic insanity, very different from simple insanity in its etiology, in its progress, and in its symptoms. The diagnosis was based, in this case, upon the general aspect, and chiefly on the nature of the mental affection, so that the paralytic symptoms, which doubtless would confirm the diagnosis when they did appear, were nevertheless not necessary to establish it. M. Parchappe, on the contrary, maintains, in this respect, the opinion of Esquirol, that in order to constitute paralytic insanity, it is necessary that paralysis should be actually present, and until it is present, the case is only one of simple insanity. But this difference between M. Parchappe and M. Jules Fabret is a circumstance of the greatest importance. Having made this explanation it will be easy for me to state, in a few words, the new opinion which I wish to see adopted. I am firmly persuaded that almost all cases of ambitious mania ought to be separated from simple insanity; but I do not agree with Bayle and M. Jules Fabret, that they should be necessarily referred to paralytic dementia, of which they may only constitute the forming stage. They ought, in my opinion, to be referred to a distinct category, under the name of congestive mania. Their relation to paralytic dementia is the same as that of ordinary insanity to simple dementia.”

The first published notice of this disease, in America, was by the late Dr. Luther V. Bell of the McLean Asylum for the Insane, Mass. : in his annual report in 1843. Dr. Bell had seen the disease in the European hospitals in 1840, and though he was not able to recall cases in that asylum previous to this period, as probable paralytics of this type, during the three years from 1840 to 1843 he had observed not less than twelve to fifteen well marked cases. This would infer its previous existence undetected. Dr. Conolly, an English writer of eminence, in his report of 1849 says, in speaking of the early somatic symptoms, the slight faltering in the speech and a little infirmity in the gait, which he considered the most prominent symptoms, that they were only discovered by those who looked for them. By this remark he evidently intended to impress the fact that they are only apparent on close examination, as he immediately afterwards gives a clear account of the psychic symptoms, and says of this early stage, "in all these cases the death-blow is struck from the first." Dr. Bell says his cases, with a single exception, were men, and that none had recovered.

In the *American Journal of the Medical Sciences* for April, 1847, eleven well marked cases are given by Dr. Pliny Earle, the present medical superintendent of the State Lunatic Asylum at Northampton, Mass., who was in 1847 the medical superintendent of the Bloomingdale Asylum, and in nine of these cases post mortem notes are given, with morbid appearances similar to those we shall hereafter describe.

The late Dr. Brigham, in an editorial in the *American Journal of Insanity* for July 1847, on paralysis peculiar to the insane, mentions Esquirol as the first to call attention to the disease, in 1805, quotes from the report of Dr. Bell, and reproduces two of Dr. Earle's cases and gives some which had come under his own observation, but does not enter into a general consideration of the disease.

The reports of all the leading hospitals for the insane, in this country, have for years recognized the existence of general paresis. The following table shows the number admitted each year into the institution at Utica:

YEARS.	ADMITTED.			DIED.		
	Men.	Women.	Total.	Men.	Women.	Total.
1849.....				4		4
1850.....	1		1	2		2
1851.....	1		1	2		2
1852.....	1	1	2		1	1
1853.....	6	1	7	4	1	5
1854.....	4	1	5	4		4
1855.....	7		7	4		4
1856.....	2		2	3		3
1857.....	9		9	3		3
1858.....	4	1	5	4		4
1859.....	5	1	6	3	2	5
1860.....	9		9	9		9
1861.....	8	1	9	10		10
1862.....	7		7	4		4
1863.....	11		11	9	1	10
1864.....	15	2	17	12		12
1865.....	21		21	12	2	14
	111	8	119	89	7	96

Dr. Joseph Workman, medical superintendent of the Provincial Asylum, Toronto, Canada, in May 1865, read a paper before the Association of Medical Superintendents of American Institutions for the Insane entitled, "Cases of insanity illustrative of the pathology of general paralysis," which was published in the *American Journal of Insanity* for July 1856, and a second paper, on the same subject, before the Association in 1858. The *American Medical Monthly* for June, 1858, contains a paper on "Paralysis Generale," by the late Dr. Ranney of New York City Lunatic Asylum, in which several cases are detailed. The *American Journal of Insanity* has reproduced several monographs published in England, France and Germany, on the subject.

Whatever difference of opinion exists as to the precise name this paretic affection should receive or as to whether it is a distinct disease or simply a complication in other recognized forms, all writers coincide in the description of the symptoms and progress, and regard the lesions of the brain, spinal cord and membranes as the cause of the paralytic phenomena. If it is only necessary, therefore, to show that a certain group of symptoms are uniformly resultant from a certain cause to establish the distinct character of a disease, this form of paralysis must be considered a disease *per se*. The development of mental disturbance, however slight, with the earliest somatic symptoms places paresis in the category of so called mental diseases. The uniform and parallel progress of the somatic and psychic symptoms and their interdependence would preclude the theory of paralysis engrafted on insanity or the converse—and this position, we conceive, is strengthened by the fact that general paresis is rarely, if ever, developed in the chronic insane. In my own experience of fifteen years, in the observance of more than five thousand insane, I have not seen a single case. Furthermore the fact that in some sections no cases of paresis occur, would add assurance to this opinion, as in the instances already cited of Montrose and Edinburgh, both old institutions.

We cannot admit that there is or can be disease of the mind, but that every case of so-called mental disease is disease of the brain, functional or organic, and the fact that the connection in life and action between mind and body is subtle and incomprehensible is not sufficient for the assumption that the mind may or can be sick. It is fair to infer that if it may be liable to disease it must be subject to death. Fortunately, however, those who would refer such a question to the domain of science, have, in the organic changes discoverable after death, a satisfactory reply. Post mortem examinations, chemistry and the microscope answer all the speculations of materialistic philosophers who would subject spirit and body alike to disease and death.

Dr. Bucknill believes "that general paresis is essentially a disease of nutrition affecting the whole nervous system, and that nerve matter, both in the vesicular and tubular portions thereof, is imperfectly produced, and that the cerebral or generative and the conducting functions are consequently interrupted." He sustains this opinion by the fact, first observed by him, that in paresis "the irritability of the muscles and the excitatory functions of the nerves are nearly lost, while in ordinary paralysis, whether dependent on lesion of the spinal cord or of the brain, these func-

tions are retained." From this he would deduce the opinion that "this disease is not located in any one portion of the cerebro-spinal axis, but consists in some morbid change, pervading the whole nervous system, and probably implicating the distal fibrils." Dr. Joseph Workman considers paresis "a distinct and essential disease of the brain," and avers that "its claims to this independent status are surely not less clear and legitimate than are those of phrenitis, hydrocephalus, delirium tremens or apoplexy." Dr. Soloman, on the essential nature of the disease, remarks: "It is a mental disease, and has all the characters pertaining thereto (insania). It is an independent form of mental disease, for it has signs, both symptomatic and anatomico-pathological, belonging exclusively to itself. These are principally mental and paralytic symptoms, going hand in hand, and being progressively developed, with a dissolved state of the cortical substance.

"Mental disease, whose expression is a disturbance in the action of the human mind, can not exist without a morbid change in the organ of mental activity, viz: the brain. In this change science must seek the cause and essential nature of the disease from an anatomico-pathological point of view.

"Two views have been entertained with respect to the essential nature of the disease, namely, the French and the German:

"1. The French view regards paresifying insanity as an inflammatory disease, arising as the result of irritation produced by repeated congestions, and causing a disorganizing inflammation. The anatomico-pathological names given by French writers to the disease refer exclusively to this theory, as, for example, Bayle calls it *meningitis chronica*; Calmeil makes it a *peri-encephalo-meningitis chronica diffusa*; Belhomme calls it *meningo-cerebritis*; while Parchappe has proposed to term it *cerebritis corticalis generalis*.

"2. The German view declares the disease to depend, as is shown by demonstrated facts, upon obstructions produced in the vascular walls (in the pia mater and cortical substance) by degeneration (hypertrophy), with their results, ischæmia and inflammation. The primary cause, therefore, is degeneration of the vascular walls. Hence proceeds derangement of the circulation, with its consequent disturbed nutrition. The secondary cause is a spreading and destructive excessive formation of connective tissue in the cortical substance, leading to the destruction of nerve-cells.

"As long-continued cerebral hyperæmia may exist without being attended with degeneration in the vessels of the pia mater, and excessive formation of connective tissue, it is clear that something more must also be present. This additional element is supposed to constitute the peculiarity of the disease, and to be of a degenerative nature.

"The diffuse periencephalitis (general paresis) presents incontestibly a striking analogy to diffuse nephritis (morbus Brightii). The former is anatomically characterized by a degeneration in the tissue of the cortical substance of the brain, destroying the nerve-tubes and nerve-cells. Clinically, it is characterized by a profound alteration in the function of the cortical substance of the brain. The latter is anatomically characterized by a degeneration of the tissue of the kidney, and by alteration in the

urinary canals and Malpighian bodies. Clinically, it is characterized by a profound change in the function of the kidneys. In both diseases we observe stages of hyperæmia, increase of volume, degeneration (softening) and atrophy.

"In the present state of science, we must lay it down that the disease consists essentially in a *degenerative process in the adventitious membrane of the vessels of the pia mater, and in the tissue connecting the elements of the cortical substance of the brain (neuro-glia), which degenerative process, in its development, causes the change of the grey cerebral cells into an inert mass.*

"When the disease has attained its climax, the use of the animal muscles is completely abolished, and the vital process is deprived of mind (anima). The disease is sometimes developed suddenly, but more generally it is insidious and slow in progress in its early stages, and nothing may be felt beyond a slight degree of general ill health, a sense of fullness in the head, or dulness and drowsiness, until the occurrence of momentary impairment of speech or slight tingling or numbness in the fingers or arms may attract serious notice. There is, however, in most cases, before these paretic premonitions, a general hyperæsthesia of the whole nervous system, producing restlessness, irritability and increased physical and mental energy, and the person works and moves more quicky than natural, is more energetic in the performance of labor or business, has better command of language, more rapid flow, and more exalted flight of thought, but at the same time he is fitful, wavering and impersistent, and though friends may not suspect insanity, they may notice a change of character, and are often pained at sudden and rude outbursts of passion, impatience and intolerance of opposition to plans. And these exhibitions may for a long time be controlled in the presence of strangers, but at length, under this increased energy and exalted idea of his capacities, he is led, without apparent cause, to change his vocation and embark in the wildest schemes and speculations, and his insanity is then fully recognized.

In some cases there is a well-marked stage of stupor and drowsiness preceding this excitement, during which the patient complains of cephalalgia, of languor and inaptitude for exertion, and of confusion of thought, loss of memory and uncontrollable disposition to sleep. During this stage there is often a vague consciousness of illness, though the character or weight of the disorder is not suspected by the patient or his friends, but as soon as the stage of exaltation and excitement supervenes, the patient feels strong and vigorous, and persists that he never was so well in his life.

This stage of drowsiness, cephalalgia and consciousness of indisposition, preceding that of excitement, we have not seen mentioned, yet we have, in personal experience, the most unquestionable evidence that many cases are thus initiated, and we believe that the non-recognition of this stage is doubtless because patients are not placed under treatment in asylums during its existence, and the family or friends, giving the history, dating the insanity only from the marked symptoms of cerebral excitement, the preceding history, involving this stage, is not obtained. Careful inquiry from those intimately associated with patients during the incipient



period, and in one instance the diary of the patient, and in another the letters written before insanity was suspected, establish this stage. There are cases also where the stage of exaltation and excitement is preceded by well-marked melancholia, with more or less confusion of thought and manner and indisposition to exercise, and others where melancholia alternates with excitement. Ordinarily, however, after the preliminary stage of hyperæsthesia and restlessness, the patient begins to develop exalted delusions about himself, his capacities, his wealth, present or prospective, and his family connections, declaring himself stronger, wiser or better than any other person. This undue and exalted idea of his own judgment leads him to talk of his plans and speculations, which, though whimsical, wild and impossible to others, are to him wise, prudent and feasible.

During the early course of the disease, the sexual desire is often increased to such a degree as to occupy the entire thoughts of the patient, and the amount of indulgence in some cases mentioned to us would seem impossible. There is also in this stage of excitement great increase of appetite and active digestion, which are not common in the early stages of ordinary mania, where the patient desires but little food and feels that he can almost do without it, while some actually despise it and appeal to the resources of a spiritual nature to sustain them. In general paresis, with all the exaltation and grand conceptions, they never desire to lessen the quantity of food, though they may be indifferent to the quality. A disposition to indulge in intoxicating drinks and in the excessive use of tobacco, is also quite general, while at the same time, in these very cases, it is not rare to find among the extravagant plans projected, the erection of churches, hospitals and asylums, generally of the most expensive character. At this stage a slight tremor of the tip of the tongue and ends of the fingers may be detected, especially after a period of quiet. As the disease advances the man rises in the scale of self-importance. He becomes immensely rich and benevolent as well, offers his check for millions, owns all the railroads, telegraph lines, &c., is about to be raised to the highest social and political positions, then attains them; he is a preacher, a doctor, a general, a president all at the same time, and not unfrequently the Saviour of the world and God himself. In a few cases this stage of exaltation is attended with one of depression, when he will whine and cry, declare his enemies are trying to steal away his wealth, but in this, as in the stage of exaltation, his appetite and love of food continue, which is not the case in melancholia.

All these manifestations may appear in a few days, or they may gradually develop during several months, increasing and remitting from time to time. At this period the somatic symptoms are usually so fully present that the disease need not be mistaken for any other form of insanity. The tremor of the tongue is now more perceptible. When about to speak, there may be observed a peculiar twitching and tremulousness of the eyelids and about the angles of the mouth, and paralysis of the tongue may be detected in a thickness of speech, as though the man was slightly intoxicated, or this paralysis may be detected only in the pronunciation of a few words, or in a drawling of speech in uttering certain words or syllables. The first stage of the disease, as may be observed, is marked by many of

the premonitory symptoms of mania, yet there are points of difference. Both forms of disease are produced by more or less disturbance of the general health. In paresis, this is more marked in connection with the function of the brain and spinal cord, as exhibited in the increased hunger and keen appetite for food, and in the excessive venereal propensity. In both, the delusive ideas are expansive, but in paresis they outstrip mania and are more incongruous and wild. In mania, there is perversion and suspicion, and false ideas seem to be generated from a false or perverted estimate of the conduct or words of others, while in paresis there is, from the first, a self-consciousness and self-assertion which prevents the patient from taking into view the conduct or words of others. His ideas and wild plans and extravagant conceptions, spring from increased cerebration and nervous energy, as in mania; but in mania the delusive state is from perverted perceptions and conceptions, and in paresis, generally, from expansive ideas touching the actual business he is engaged in. In developing mania, the patient fears opposition and suspects persons are plotting to thwart him or outrival him. The parietic has no such fears. His plans and schemes are too vast for competition, and his resources of money, wisdom, judgment, friends, social and political standing are equal to the magnitude of the projects entertained. As for himself, he is the center of all, and annihilates all opposition with a word. The delusions of the parietic are also progressive, while those of mania are not. The maniac may expand as to the number, but not as to quality.

In paresis, the poor man is first rich by owning a few hundreds, then thousands, then millions, then lands without limit. The politician is successful and advances steadily till he exceeds the grandest climax of human ambition; then he is Christ and God, and, as in one case which I present, proclaims himself the father and grandfather of God. This we know is not found in mania. In what the French have called "ambitious mania," which has by some been confounded with paresis, we find the most extravagant delusions as to wealth and power, but these have been reached at once, and associated with them are no indications of paresis. From such supposed parietics recoveries are sometimes reported, and in other cases remissions or intervals in paresis are reported as recoveries, as recently, by M. Piñel.

Sooner or later a maniacal paroxysm is developed. Often in a few days, at other times weeks and months after the first symptoms appear. After this all the symptoms are aggravated. The tongue on protrusion is not only tremulous, but, if well protruded, is jerked back suddenly, apparently and really against the effort of the patient to continue the protrusion. There is noticeable dullness and want of animation in the whole physiognomy of the face. The eyes are generally more prominent than normal. If the arm is extended, a tremor of the extremities of the fingers and often of the whole hand may be detected. If the patient writes, the letters will be angular and scratchy and connecting words and parts of sentences omitted. He has more or less difficulty in all detailed actions, such as picking up small articles with his fingers, buttoning his clothes, putting his fingers in a vest pocket,—though in the general movement of the arms this may not so readily appear. His walk is slow, irregular and uncertain, and one foot may be lifted higher than the other or drop harder on the floor, or drag

somewhat behind, or the walk may be a sort of straddling stride, something like the walk of a sailor. The restlessness characterizing the earlier period of the malady may abate and the patient become quiet in manner, and occupy himself in the contemplation of all the good things surrounding him—for in this stage they are not only well pleased with themselves, but almost universally with everything about them. Their clothing and bedding are of the finest material. They have the most sumptuous dinners, the greatest number of the most charming friends, and the best wives and children, and often scores and hundreds of each.

After longer or shorter periods repeated attacks of epileptiform seizures supervene, each leaving the patient more and more prostrated and more feeble in mind, and not unfrequently in one of these attacks, the scene closes in a convulsion or hemorrhagic apoplexy, but more commonly death ensues from gradually accumulated serum within the arachnoid, producing coma.

On the diagnosis of paresis, Dr. Bucknell remarks :

“The diagnosis of general paralysis is practically of the most facile sort, although it is not easy to describe in words the slight but pathognomonic changes which speak so forcibly to the practiced eye and ear of the observer. The one diagnostic symptom of the early stage of paralysis, is the modification of articulation. This is neither stammering nor hesitation of speech. It more closely resembles the thickness of speech observable in a drunken man. It depends upon a loss of power over the co-ordinate action of the muscles of vocal articulation. In many instances the speech of the early paralytic is fluent and clear, except in the pronunciation of certain words, or sequences of words, which require the neat and precise action of the muscles of speech. Words composed of vocal sounds connected by single consonants are articulated with correctness ; but words composed of numerous consonants, with few vocalic sounds, are articulated in a shuffled manner, which is perfectly characteristic. The patient may even possess the power of articulating these words correctly, if he purposely attempts to do so ; but if the examiner holds him in conversation for a few minutes, the ear will infallibly detect the slight but fatal symptom of incurable disease.”

It is unnecessary to discuss further this branch of the subject, but I propose to present a few cases practically illustrative of the symptomatology, psychic and somatic, of the disease.

The following cases illustrate in a very marked degree the early stage, the expansive ideas, the excessive sexual appetite and indulgence in intoxicating beverages.

#### *Case First.*

G— ; aged 56 ; married twice ; father of six children ; temperate habits ; admitted February, 1864. On admission he had the somatic symptoms of speech, twitching at angles of mouth and uncertainty of walk well marked ; and his ideas were of the most wild and expansive kind. He wanted to buy the farms on either side of the Hudson for a number of miles ; to build a horse railroad from Hoosick Falls to Troy ; to dam the Hudson and make a great water privilege ; to build a large free church, and to

make large donations of money to many of his friends. When told his means were too limited, he replied: "Money! Why I am all money—can command millions and issue checks whenever I please, and they must be cashed. My name is good everywhere."

On his way to the Asylum he tried to kiss every woman he met, and proclaimed his intention to marry all the women in the world, and raise fifty million of children. Offered also to take people's "insides" out, or to take their heads off and replace them by new and much better ones. Thought also he could raise the dead and kill people and bring them to life again. Had also the delusion that he could make a flying machine, by which he could sail through the air, propelled by perpetual motion. Said that by eating half a ton of coal and drinking a barrel of water he could "steam up" (to use his own words), "and go to Heaven and bid God good morning before breakfast." He ate enormously of the most nutritious food.

All these symptoms were developed suddenly about four months prior to his being brought to the Asylum, and were initiated by a great debauch in New York; a thing he had never been guilty of before. Such is the history obtained from a friend of the patient who brought him to the Asylum. His wife visited him about three weeks after admission and gave the following additional history:

She said her husband's health had been gradually failing for more than a year before the attack. He had complained of languor, severe pains in his head, and unusual heat of body generally. At times he appeared much abstracted; and at other times irritable, excitable and sleepless. And this condition was nearly always followed by great drowsiness, so that he would often fall asleep while people were talking to him. During this time attended to his business steadily and successfully. During all this time was very anxious. After five or six months his restlessness, nervousness and irritability increased, and he had what he called "shocks" about his throat, inducing a sensation as though some one caught him by the throat. In one of these "shocks" he lost his voice, and ever afterwards had a "trembling" in speech. At this time he had a constant desire to micturate; he increased in flesh rapidly.

His appetite was keen and voracious. His hands and fingers became quite tremulous, and he walked with feet wide apart and faster than usual, as though he was balancing himself.

In the spring of 1863, from being cautious and prudent, became extravagant in ideas, and proposed expensive and unnecessary changes about the house. From being kind and genial in his family, became selfish and indifferent. Up to this time still attended prudently and cautiously to business. His head ache continued. Occasional attacks of diarrhoea came on which relieved his head ache and left him more composed and rational.

In May, and subsequently, he had hallucinations of sight, whether his eyes were opened or closed. He saw beautiful moons, stars, flashes of light, &c. These he continued to describe up to October. During the spring and summer was subject to night sweats, often wetting his shirt and sheets thoroughly. Towards the fall his sexual appetite diminished very much, although he talked about women in a way to indicate the opposite of this.

This, it will be seen, brings us to the time at which the final explosion took place, and at which the history first detailed commences.

This case has run and is now running the usual course. He has continued to maintain the same general range of delusions with little variation, except that as he becomes more demented he has fewer notions, and the few he has are less active.

The somatic paresis is also gradually increasing and he has had four or five slight apoplectic seizures, rendering him unconscious for a short time at each attack, and leaving more or less paralysis of left hand and side. He appears now to have arrived at the *third* stage of the disease, but has not yet passed through the period of excitement common to the *second* stage; and we apprehend he may gradually fail and the case terminate without excitement. He eats largely and voraciously; he sleeps well and is very fat. His bowels are very torpid and require laxatives every week at least.

#### *Case Second.*

J—; male; aged 53; married; 7 children; merchant; temperate habits; admitted, November, 1864. A sister once had a maniacal attack, and a cousin was insane.

Has suffered from chronic catarrh for sixteen years. Has frequently complained, during the past year, of sensation of fatigue, difficulty of concentrated mental effort, loss of memory, particularly of recent events, failure of eyesight (amaurosis?), occasional rigors (nervous?). Three weeks ago remarked that he must have had a stroke of palsy in the night, as he had awaked cold, clammy, feeble, with a stiffness in the left side, some difficulty of articulation, and inability to "collect his thoughts."

This condition was not such as to attract any special notice until the 3d inst. At one o'clock in the morning he was wakeful and restless, and on being interrogated, informed his wife that he was contemplating the building of a very large hotel, which he would commence in the morning. At 3, a. m., complained of inability to sleep, said he would take a walk and left the house. Was discovered some hours later making bargains for the contemplated edifice; drew check on bank for \$2,500, and offered to bet the amount on either side in the impending election. The same day attended a political meeting, and on returning home at night asserted he was the richest man in the county and the State; he was a Governor, President, Saint, King, Washington, a God. Was seen by his physician the next evening when he was maniacal, with quick, rapid pulse, flushed face, hot head, etc. On being restrained, became violent and destructive, and was bound and shackled. Was bled 16 oz., cathartics given, blistered on nucha, and then ordered potassii iodidi, gr. v, ter in die. On Thursday, in a struggle with his attendant at home, dislocated from his spine the 6th, 7th and 8th ribs. Brought to the asylum to-day, and has cough, difficult respiration and bloody expectoration. November 22d. Quiet most of the night. Visited this morning by wife and friends. At noon refused food, swearing he would eat no more here. Threw off his coat and threatened to slay any one who opposed his departure. 23d. Last night destroyed his mattress; noisy; talking in exalted, incoherent manner of his wealth, power and grandeur. 29th. Quiet since last note, but incoherent and exalted in his

views. December 2d. Says Governor S. has been to see him; that he (patient) has been twice killed; that this institution is a d—d place which he built and carries on as he pleases. Accuses a fellow-patient with having destroyed his property, and wants it charged to him. Takes anodynes. 8th. Every afternoon at 2 o'clock his face is flushed, head hot, and he is drowsy; this followed by reaction, restlessness and loquacity. 24th says the world will, by his command, be destroyed next Saturday. 30th. visited by wife. Soon after her departure he became noisy and abusive of his wife. Last night he was up, kicking the door, throwing bed about the room, etc. January 25th, 1865. Delusions unchanged; paresis of muscular system, tongue, etc., increasing; appetite voracious; has no realization of his condition. February 27th. Quiet, but ideas the same. April 22d. Quiet since last note; at times reads or plays dominoes. July 7th. Discharged "unimproved," by request of friends.

#### *Case Third.*

B—; aged 32; married; no children; real estate broker; common education; a great smoker from boyhood, and for several years a *bon vivant*; was admitted November, 1864; had been stout and robust from childhood; was regarded as somewhat eccentric; noted for his irritable, capricious and emotional temperament. At the age of puberty he fell into the habit of self-abuse, and continued it more or less up to his marriage, two years before his admission; although he was known to have frequently indulged in sexual excesses during this time. Two years before admission he was married to a young woman 14 years his junior, and during the honeymoon he made many presents to her and her friends, and spoke much and in glowing terms of his future business prospects.

His friends thought him a little peculiar, but never suspected his mental capacity. He managed his own business prudently, but was lavish and extravagant in his expenditures for domestic purposes.

In nine months more he became gradually restless and nervous, and engaged in petroleum speculations, and sundry other more extravagant things. About this time became very suspicious of his wife, and insisted on having her with him wherever he went. Began at this time also to exhibit all the usual parietic symptoms of the muscular system.

His wife informed us that his sexual appetite was beyond all bounds for a year after marriage, insisting in cohabiting three, four, or more times, even eight, in succession; but part of this time he could not contain himself until intromission was effected. The more incompetent he became in this respect, the more excessive his appetite appeared to grow, and his wife was obliged to leave him and go to her friends.

On admission he was greatly demented and scarcely able to walk. His speech was very slow and indistinct, and his appetite large. The few ideas he yet had were gloomy and depressing; he thought he was rotten inside, or transformed. Soon after he came he refused food under the delusion he was dead. Was very helpless and filthy, addicted to self-abuse and had to be restrained. Under generous diet, with ale and cod liver oil, he improved in health and began to grow fat. He is now much demented.

This case well exhibits the peculiar temperament most liable to the dis-

ease, its early stage, the exaltation and ideas of wealth, the excessive appetite for food and spirits and the gradual somatic paresis. It also shows a great change from exaltation to depression, and also excessive venereal appetite. How much these excesses may have had to do with the induction of the disease, we would not venture to conjecture, but that they may have been sufficient there can be little doubt.

*Case Four.*

W—; admitted April 1861; aged 48; married; nine children; produce, cattle and real estate dealer; strictly temperate; of quiet domestic habits; a strong, vigorous man; occasionally has suffered from sick headache. Two years ago had an attack of inflammatory rheumatism after which had sick headache. Several months before admission suffered considerably from rheumatism, but was unusually active and speculative. Friends did not suspect insanity but were anxious about him, as they feared he would go too far, and lose his property, which was large. He talked well but extravagantly; was early and late at business; sleepless; ate largely and in harmony with his increased energy and work. Became excessive in sexual indulgence, and made overtures to his servants which were so antagonistic to his character that they thought he had been drinking. This excitement steadily increased, and from being reticent about his business affairs, he began to talk them to every one, boasted of his wealth and influence and proposed the most extravagant speculations. One day he burned a lot of bank notes to show his indifference to small things, and when the act was characterized as foolish, and that no one would gain thereby, he replied, "yes they were greenbacks, and the government will gain by it, and I can afford it." His physician says that at this time his rheumatism disappeared, and he suddenly developed furious mania. His delusions of wealth and power were of the most extravagant character. He was taken to an asylum, but paresis was not suspected. Soon afterwards he was brought here. The parietic symptoms were all well marked. He entered the ward quietly, but subsequently proposed to leave, and when again told that he was to remain he declared he would take the house down. In a few days he became maniacal, and this state subsiding, he had several epileptiform seizures. After this he was comparatively quiet, but the somatic symptoms increased, and his delusions became more and more extravagant. He had purchased California, Texas and Illinois, which he was about to plant in onions. Had 1,000 horses which increased to 40,000. Had five millions of men working for him. Had constructed a ship canal from Chicago to San Francisco, and contemplated carting the Rocky Mountains into the Atlantic ocean to make a bed for a railroad to England; the removal of the asylum to his home for a residence, which at one time he would propose to draw with two mules, at another he would draw it himself, and start the next morning; that he would marry the Queen and Jenny Lind and set up a harem, and out rival Brigham Young in wives and children.

He had epileptiform attacks which increased in frequency, and at length, seven months after admission he died apoplectic after a series of these convulsions.

*Case Five.*

D—; admitted August 1864; aged 57; married; three children; shoemaker; good habits.

This case came on gradually much as the others, but after the appearance of the marked somatic symptoms, progressed rapidly, and he died nine months after the attack, entirely paralysed. The striking feature in this case was the occurrence of priapism for several weeks before death, and the almost constant discharge of seminal fluid diluted with mucus.

The following cases illustrate the intervals or lulls in the progress of the disease which sometimes occur, and the consciousness of illness.

*Case Six.*

C—; admitted January 1857; aged 34; married; physician; intemperate; smokes and chews; academic education; not religious; native of New York. Hereditary; paternal grandmother insane.

Was a healthy, intelligent child, but was indulged at home to his own detriment. At the age of 18 was an egotist, a profligate and a spendthrift. Graduated in medicine at the age of 22, but did not succeed rapidly in practice. Was infidel in his notions and low in his associations.

Letters written by patient August 1855, show mental derangement; an exalted affection for his family, wild notions of fame, great boastings and slight disconnectedness of ideas. Subsequent to this, nothing learned as to his condition until the spring of the following year, when he became dejected, remorseful, accusing himself; gave up drinking; became taciturn, seclusive, meditated suicide (melancholia). At present appears in good physical condition; eats heartily and sleeps fairly. Has all the somatic symptoms of paresis.

Condition in April; that of a paretic dement, is taciturn, filthy, &c. Between this and November he had one or two maniacal paroxysms with great prostration. During this period tonics and stimulants were administered.

In November he began to improve, and by the 15th of the following June he was removed by his friends, they thinking him well, and went home to resume his practice as a physician. It is proper here to state that we informed the friends that this was but a treacherous lull in the disease, and that its return at no distant day was certain.

Patient was re-admitted on the 20th of May, 1859, (18 months). His history during his absence is briefly this. During the summer he was indolent, taciturn, irritable, apparently disheartened at having nothing to do. In the fall of 1858 he visited this asylum, and applied for the situation of assistant physician. At that time was thought by his friends to be well enough to attend to professional duties if under some supervision, but not well enough to conduct a general practice. About three months ago, his irritability left him, he became exalted, held delusions of wealth. Is now exhilarated and excited.

June 10. Is very filthy, chewing rags and shavings from spitboxes. July 26. Two days ago, first symptoms of simple sanguineous cyst (hæmatoma auris) occurred—effusion in three days and ridges and depressions of ear quite obliterated. July 31. Tumor fluctuating. There has



been little or no pain, or any modification of mental state since ear trouble began. Patient's conversation seldom turns upon himself, and he apologizes when he speaks of himself as "the best physician in the world," excusing his selfishness, &c. A few days ago bit his attendant in the arm. Is destructive, filthy, occasionally requiring restraint. Tumefaction of ear not abated but rather augmented. August 2. Filthy, smearing wall with fœces and defiling room. August 8. Asserts that he is the "grandfather of God Almighty. That dame nature is his wife—that he formed her out of the most precious materials, jewels, diamonds, butterfly's wings, &c., which he made with a forty horse power." Says he "died once of bilious cholera—was buried for fifteen years, corruption taking place, but that an angel came down and resurrected him." August 14. Has slept badly for some time past. Is up and about room much of the night; when spoken to of his wakefulness, insists that he has the most perfect repose—that it seems as though he could not sleep enough or keep his eyes open a minute at night. August 29. Appears feeble this morning; slept none last night, was delirious and noisy. September 6. Up to-day. Talks incoherently and incessantly—is destructive of clothing. January 7, 1860. Another maniacal paroxysm. 11th. Occasional attacks of diarrhœa. February 17. Excitement abated—emaciating. Takes ol. jec. asal; is destructive. March 24. Complains of itching of face, which he picks and wounds unless he wears camisole. Emaciates in spite of ol. jec. During April no special change. May 3. For past two months there has been a remission in the maniacal excitement—conversation more rational—absence of characteristic delusions but mind more feeble; manner listless; habits still filthy at times; perversion of taste; no control over sphincters; emaciating; right lower jaw tumefied; asks to go home. May 8. While sitting up this a. m. had epileptiform seizure, resulting in slight facial paralysis. Is more feeble and failing; ordered special night attendant. May 10. Died suddenly this a. m.

*Case Seventh.*

B—, admitted December, 1863, with all the symptoms of paresis well marked. In the early part of 1863, for four or five months, was drowsy and dull, but these symptoms did not then attract attention. When the stage of exaltation developed, he left home, entered into speculative schemes and lost property. Subsequently went to a neighboring State and enlisted in the cavalry service. His wife found out his whereabouts, and endeavored to obtain his discharge on the plea of insanity, but failed; the officer insisting that he was at times only under the influence of intoxicating drinks. In a short time, however, he became noisy, incoherent, and so manifestly insane, that he was discharged and immediately brought home.

On admission all the somatic symptoms were fully manifest. His appetite was keen and large, digestion good, and he presented the appearance of a man in full health. The tremor of tongue and fingers was very perceptible and his speech characteristic. He had exalted delusions of wealth and power. The delusions soon subsided and the parietic symptoms soon receded. He spoke of his former state and present condition, his difficulty of speech, uncertainty of gait, loss of memory and of power of attention at times, and in power of thinking. He, however, did not manifest an appre-

ciation of his danger, and this could not be impressed upon him. He was confident and hopeful—went out in the garden to work in the spring of 1864, and though we could detect by examination the continuance of the paresis, to ordinary observers he seemed well. His conversation was rational. He eloped in May, and on reaching home seemed so well that his friends wished him to remain. He worked through the summer, and though he continued muscular and stout in appearance, he was not able to accomplish a great deal. In the fall all the somatic symptoms began to increase. He at length requested his wife to secure his return to the asylum, and while arrangements were being made, he left home and came here and begged to be received at once, stating that he “was sick and losing his mind again.” Since then he has had several epileptiform seizures, and is now so paralysed that he is unable to dress or undress. He is serene and comfortable—dementing—yet he is conscious of his condition, and has had other patients write letters for him in which he informs his wife of his progressive illness and loss of mind.

We have as yet little positive knowledge touching the predisposing or prominent cause or causes of this terrible disease. In many cases there is strong hereditary tendency to other forms of insanity or other cerebral diseases, and most of these patients have been nervous or excitable people, manifesting in health great buoyancy, vivacity and mobility of mind.

As a prominent cause it was thought formerly that intemperance stood foremost, but it is found that some of the most marked cases were through life of the most strictly temperate habits, up to the period of attack, and that, in such cases, intemperance was only a result of the disease. Some, however, have been intemperate for years. Two of those admitted this year were not only intemperate from boyhood, but always exceedingly susceptible to the influence of ardent spirits, so that in their best state of health a comparatively small quantity would set them wild and almost delirious.

We have nothing to offer in the way of treatment beyond the general statement that this class require a large amount of nutritious food in all stages of the disease. Active treatment, after the disease is fully developed, blisters, setons or low diet only procure discomfort and hasten death. The treatment should be sustaining in the sense of nourishing and sustaining the system and securing sleep.

On the pathology much has been written, but with little satisfaction. It is true that post mortem examinations, under the naked eye and under the microscope of distinguished observers, exhibit much that is interesting to the pathologist; yet there is no pathological condition sufficiently grave and constant to account for the manifold symptoms. Moreover, the conditions we are mainly limited to, in our examinations, are not the exponents of the early symptoms, but the termination of a long chain of disordered action, which, though revealing ample cause of death, does not enable us, by any process of inductive reasoning, to reach a satisfactory explanation of the conditions inducing the disease.

I have not followed the general rule of dividing into stages, because I have not sought to present an essay on the disease, but simply, as already

stated, to give an outline and sketch, with some cases which might induce more careful observance of the early symptoms, by the general profession, and stimulate the study of the disease, that one might, if possible, solve the question as to the conditions in which it has its origin. Until this is done it will remain, as it is now, the opprobrium of the profession; for after actual lesion of structure has taken place, as indicated in the paralysis of the tongue, we cannot hope to do more than palliate and delay, or smooth the inevitable road to death.

I have not dwelt on differential diagnosis, though I took occasion, while noticing the early stage, to point out some distinctions between the developing symptoms of mania and paresis. This, I think, is the only stage in which the two affections are likely to be confounded. The early stage, and especially the premonitory symptoms, should receive study and observation. There is, unfortunately, no want of material to perfect the history and character of the disease, and its fatal issue after the parietic manifestations.

The diseases with which paresis has been confounded are apoplexy, chronic alcoholism, and paralysis from muscular atrophy, and I cannot do better than present the remarks of Salomon upon this point:

"1. *Apoplexy*.—In a slighter attack of apoplexy, where the paralysis affects the tongue, it is exclusively or predominatingly unilateral, on which account the tongue turns to one side when it is protruded. Hemiplegia, paraplegia, &c., present not the slightest similarity to general paresis, for in such cases the paralysis is complete in the parts of the body affected, and moreover is partial and not general.

"2. *Alcoholismus Chronicus*.—General paresis has almost invariably been confounded with this toxic disease. Even in the present day French writers especially confound these diseases, in consequence of insufficient acquaintance with chronic alcoholism.

"The group of symptoms included under the denomination dementia paralytica, belongs essentially to paresifying insanity, but it may also be met with in chronic alcoholism, when the latter has attained a higher degree of development. A man may arrive at dementia in many ways; dementia, with bodily paralysis, he may reach especially through general paresis or chronic alcoholism. When the patient has already reached the goal, it may often be difficult to say immediately, from the existing symptoms, in what way he has attained to it; but when information is afforded as to the cause of the disease, the decision is as easy as it is certain.

"The principal feature of the differential diagnosis is to be found in the dissimilar starting-points of the diseases. General paresis proceeds from a morbid process in the fine membranes of the brain; chronic alcoholism from a general intoxication. In the former case the physical symptoms occupy the first place; the degeneration of the mind tends to produce that of the body. In the latter, the paralytic symptoms are the first; the general intoxication of the body tends to the degeneration of the mind. The dissimilar etiological source of the diseases differentiates them in a decided manner. A person who has indulged in an excessive use of brandy at length becomes poisoned, and, in consequence thereof, becomes the subject of chronic alcoholism, but never of paresifying insanity. If he has, at the

same time, indulged in enervating excesses, particularly in those of a sexual character, he may, in addition to his chronic alcoholism, acquire general paresis.

"3. *Paralysis from Muscular Atrophy*.—This disease has been confounded with paresifying insanity. If this mistake is still made, it is attributable to deficient scientific knowledge in the physician. The diseases have this in common, that in both progressive paralysis proceeding from the muscular system occurs. In other respects they are wholly dissimilar. In the one the seat of the disease is in the brain; in the other it is in the muscles. Paretic patients may, under the influence of delirium, employ their muscles in a very violent manner; such a patient may dash in pieces the door of the room in which he is confined. A person suffering from paralysis from muscular atrophy does not rave, and, in consequence of the degenerated state of his muscles, cannot be violent."

He might have added that in paresis, even in the third stage, after a period of days and weeks of the most helpless paralysis, patients some times so far apparently improve as to get up and walk about, and occasionally suffer a maniacal paroxysm, whereas in paralysis from muscular atrophy no remissions occur.

Dr. Saloman describes the disease under four stages:

1. The stage of mental alteration.
2. The stage of mental alienation.
3. The stage of dementia.
4. The stage of amentia—paralysis of mind—complete dementia.

This is perhaps as acceptable as any, however, I have not always found it easy to mark the stages as arbitrarily as these divisions would demand.

There is unquestionably a stage of alteration in which the character, habits and physical condition are abnormal, and in many cases the development of mania is sudden, but in others the transition is gradual from a changed state, not of a marked character, to an exalted or delusive state of mind and increased energy of body, and this state merges slowly into one of pronounced paresis; sometimes the somatic symptoms, and at other times the psychic symptoms first appearing. Now these latter cases indicate the true character of the disease—its progressive nature; that it is not general paralysis, but paresis; a paresifying disease; an incomplete but constantly increasing paralysis. The cases coming on thus gradually, run a more regular course and live longer than others.

I shall present a few additional cases, observed in the asylum at Utica, with post mortem notes, with the pathological views entertained by some of the most prominent and distinguished writers and observers.

#### *Case One.*

J. D., cabinet-maker, aged 38, of intemperate habits, entered the asylum on the 18th July, 1854. He had then been insane for six months; "had been strange in his appearance and talked foolishly," had manifested exalted delusions respecting property, and had an impediment in his speech. When admitted was, apparently, in good physical health, was rapid and skillful at his handicraft, which he pursued for several months in the asylum

workshop. During the fall he was attacked with epileptiform convulsions, accompanied by maniacal excitement. One of these attacks was attended by hæmatoma auris. The paroxysms subsiding, left him with marked paralysis of the lower extremities, greater difficulty of speech and deglutition, and more prominent delusions of wealth, power, and happiness. By the spring of 1855 he had so far improved that he could dress himself and take out-door walks. The following summer he emaciated, but was free from maniacal attacks. With winter the paroxysms returned, but the excitement was of transient character, and the intervals characterized by greater mental and motor impairment. In the spring of 1857 the stage of dementia was reached, and from this time until his death in June 1858, he was bed-ridden, and presented the usual characteristics of the concluding epoch of this disease. It is a curious circumstance, that on the day preceding death, hæmatoma auris again occurred, and this time in both ears; the cysts forming simultaneously and within a few hours.

*Head and spinal cord examined.*—The white and grey substance of the hemisphere generally presented a healthy appearance. There was some clear serum in the lateral ventricles, and a considerable collection also of serous fluid, strongly tinged with blood, in the cavity of the spinal sheath. The bloody exudation here had the appearance of a *post-mortem* transudation. The meninges of the spinal cord were also stained of a reddish hue, apparently from the same cause. Otherwise, the meninges, both of the brain and spinal cord, were natural.

There was a very remarkable induration of the substance of the tuber annulare, on each side of the median line, which could be easily traced by the fingers, and even resisted distinctly the edge of the knife. This induration was not situated in the superficial layer of transverse fibres, forming the pons Varolii, but deep in the substance of the tuber annulare, and occupied precisely the track of the anterior columns of the cord, as they pass upwards from the medulla oblongata towards the crura cerebri. There was a similar induration of the two olivary bodies, even more strongly marked than that of the tuber annulare. The anterior pyramids were not indurated to any appreciable extent, and the remainder of the medulla oblongata and spinal cord were unaltered in consistency and texture. The induration of the nervous matter was not accompanied by any fibrinous or purulent effusion, or by any appearance of an inflammatory character. Microscopic examination of the parts above mentioned, showed the natural elements of the nervous substance, both white and grey, presenting a normal appearance. In the substance of the tuber annulare there were some fatty granules and oil drops, but not very abundant. Weight of brain was two pound and ten ounces.

#### *Case Second.*

J. C., music teacher, aged 34, for several years past had been intemperate and had practiced vicious habits. Admitted to asylum July 14, 1856. Had then been six months insane. Patient entered the ward for convalescents, where he behaved with propriety. He soon evinced inordinate eating propensities, and laughed and talked boisterously, engaged eagerly in the musical entertainments and amusements of the patients, rarely alluded to

his delusions in conversation, but filled letters with extravagant descriptions of his wealth and future expectations; was very particular as to his dress and personal appearance, and often surveyed himself complacently in the mirror. He soon exhibited slowness of utterance and unsteadiness of gait. He generally occupied a seat with the choir in the chapel, but found much difficulty in applying words to music. In October '57, fifteen months after admission, he had an epileptiform convulsion. These seizures recurred at short intervals, and were attended by transient excitement or bewilderment. The ideas became more expansive, and the paralytic phenomena more noticeable. By August, '58, had become quite demented. In September, was unable to walk, lost control over sphincters, was deficient in power and consentaneous action in upper extremities. His appetite was good. During the following month he emaciated, and was singularly wakeful. On the 2d of November he appeared brighter, attempted to converse in a lively way, called for a bottle of wine, exhibited less difficulty of articulation. During the morning of the 2d, appeared much as on the 3d—he talked, ate well, and his pulse was of good strength. Later in the day he had an epileptiform seizure, with slight muscular tremors and contraction of extremities and about mouth, face drawn down towards left shoulder, pupils unaffected. The following day his condition was the same—mouth firmly shut, eyes fixed and staring. He died on the 5th.

*Examination of brain.*—The entire brain has a strongly marked dusky or cineritious look, not owing to any venous congestion, but apparently due to a change in the color of the brain substance. There is no unnatural appearance about the meninges. There is a little more serum in the ventricles than usual, but not enough to exert any serious impression on the brain. The consistency of the external portions of the encephalon is natural, except in the olivary bodies on each side the medulla oblongata, and in the tuber annulare on each side of the median line. At these situations there is a well marked hardening, rather less pronounced than in *case one*, but still very distinct. On cutting open these parts there is, however, no unnatural appearance appreciable to the eye, and the microscopic anatomy is also normal so far as can be ascertained; no fatty degeneration, no morbid growth, no perceptible alteration of nerve-fibres or cells. There is no hardening of anterior pyramids as in *case one*. The hardening in tuber annulare is not superficial, but deep-seated. In the cerebellum the distinction in color between the inner and outer layers of grey substance is exceedingly well marked, but microscopic appearances here, also present nothing abnormal. White substance both of cerebrum and cerebellum generally, natural in color and consistency, but there is some softening (not excessive) of fornix only.

#### *Case Three.*

W. M., aged 41, merchant, temperate habits. Admitted October 18, 1857. One year before, while in Germany, he began to make improper purchases of goods, in selling was careless as to security for payment, and speedily involved himself in financial ruin. Had a period of depression lasting two months, during which he drank to the extent of four or five bottles of champagne daily. Was careless in dress, wandered about from

place to place, and was at last sent to the asylum at Wurtemberg. There he remained four months and left "unimproved." Two months ago he came to America, and has since been quiet, free from excitement, indifferent to his family and himself. Has manifested gradual impairment of speech, and his gait has at times been feeble and paralytic. He has, however, had no convulsion or paroxysm, and general health has been well sustained. The delusions are of an exalted nature; he desires to return to Germany where an inheritance of *millions* awaits him, and will start off by day or by night to take the nearest road to his Germany, which he thinks a few hours travel on foot will enable him to reach. His present state is one of paralytic dementia. He manifests a feeling of great comfort, but has no appreciation of distance or time, and his memory is impaired. The left pupil is more dilated than the right, the left side is weaker, and he grasps more feebly with the left hand. Soon after entering the asylum he was seized with epileptiform convulsions. These recurred from time to time, and were accompanied rather by transient cerebral erethism than by true maniacal excitement. He gradually sank into a condition of more profound mental degradation and paralysis, and died in September, 1859.

*Examination of brain and spinal cord 72 hours after death.* Slight putrefactive odor. Superficial redness over lateral parts of middle lobes of both cerebral hemispheres—most marked on left side. This redness apparently due to a *post mortem* settling of blood in the parts. No coagula or other unusual exudations about surface of brain. Arachnoid of natural moisture and polish; no marked opacity or thickening anywhere; brain everywhere firm to the touch externally, excepting just at junction of middle and posterior lobes; here it is a little soft and yielding. Under surface of brain, has a more distinctly putrefactive odor, and projecting portions of anterior and middle lobes inferiorly, have a greenish slate color. Vessels of pia mater, internal carotids, and other arteries at base of brain, have a natural appearance. On slicing brain from above downward, its cut surface has a very distinct *dusky* or *slaty* hue, which becomes lighter and more fresh colored after a few moments exposure to the air. Substance of cortical medullary portion quite firm to touch, *remarkably so considering* the commencing stage of putrefaction. Corpora striata, optic thalami, and other central parts of brain not altered in consistency or external appearance. In *corpora striata* and *optic thalami*, on both sides, many of the minute arteries and capillary blood-vessels had undergone advanced fatty degeneration. This degeneration was confined to isolated spots and streaks in the vessel, and many of the vessels were entirely free from it for a considerable part of their length, but it was still very abundant in the parts above mentioned. Fatty degeneration of the blood-vessels was not found in any other situation, though it was looked for in the hemispheres, tuber annulare, olivary bodies, crura cerebri, cerebellum and spinal cord.

Tuber annulare very firm in consistency, as were also the olivary bodies. Corpora pyramidalia, rest of medulla oblongata and whole of cerebellum rather soft.

Spinal cord, pia matter, and surface of cord natural in cervical portion, but of a deep permanent dark *slaty* or *blackish* color in dorsal portion, particularly on posterior aspect. In lumbar portion dark red posteriorly, as if

from *post mortem* settling of blood. No thickening or exudation. Anterior surface of cord tolerably healthy in appearance. Cord, divided transversely in thin sections throughout its entire length, showed no unnatural appearance, either to naked eye or to microscopic examination.

*Case Fourth.*

J. McG., aged 29, mason, habits intemperate. Was observed to be confused in his manner and unable to apply himself to his work, in March, 1861. Had since grown steadily worse, but without at any time manifesting excitement. There is no account of a convulsive attack, although his mouth on one side is drawn down. When admitted to the asylum, September 9, 1861, he was demented, indifferent to matters of interest, had a pleased, self-satisfied expression, a hesitancy in his speech, and defective co-ordination of movements. This state continued without notable change until September of the following year, when a slight, transient paroxysm of excitement ushered in a state of greater mental and motor impairment. From this time until his death, February 9, 1863, frequent epileptiform seizures occurred and were followed by more or less profound stupor and paralysis.

*Post-mortem examination of the brain.*—There was evident thickening of the dura mater, as compared with the dura mater of a healthy brain examined at the same time. Thickening and opacity of the arachnoid everywhere existed, but especially distinct where the membrane was stretched over the anfractuositities of the cerebral hemispheres. On raising, or attempting to raise the arachnoid from the brain, it appeared unusually adherent, and the cerebral substance was lacerated and clung to the membrane. There was an abnormal degree of congestion of the superficial vessels, and a marked injection of the vessels of the pia mater. The consistence of the brain seemed natural.

The vessels of the pia mater were very carefully examined with the microscope, and in order to appreciate more accurately the microscopical appearances, the brain of a sane person who died of phthisis pulmonalis was procured for the sake of comparison. Repeated examinations of the arteries, veins and capillaries from the pia mater, and the superficial layers of grey matter of the brain, failed to discover the hypertrophy of the connective tissue which Wedl describes. The obliteration of the capillaries and their change into corresponding bundles of fiber could not be detected, nor any approach to such change. The fatty and calcareous degeneration of the capillaries was not nearly so extensive as in the case of the phthisis patient, whose cerebral functions to the last day of his life were unimpaired. The changes which Wedl describes as affecting the nerve-cells and fibers, this examination did not confirm. There did not seem to be any abnormal appearance in the cerebral tissue.

*Case Fifth.*

T. C.—Aged 35; married; five children; tanner; common education; not religious; temperate; Irishman; admitted 1863. Four months prior to this time, appeared well and attended his work regularly. The tannery in which he worked burned down, and he worked very hard to save stock



and became greatly excited. Soon after he was depressed, gloomy and taciturn. Two months later had an epileptiform seizure, which left him nearly blind and unable to speak distinctly. In this condition he was brought to the asylum, and on examination it was found that all the somatic symptoms of paresis were present, though he did not have the characteristic restlessness and exalted notions, but was, on the contrary, fearful and melancholy.

Only the head was examined. Dura mater was adherent at apex along both sides of falx. Inside of it of yellowish red color; within arachnoid a large quantity of serum. Pia mater of dirty creamy color. Whole of brain substance softened throughout, and in some places more than in others. Cortical portions, jelly-like in consistence. Puncta vasculosa not noticeable. Thalami optici, corpora striata, falx and crura cerebri, all softened. Optic commissure and proximal end of optic nerves of creamy consistence.

#### *Case Sixth.*

W. G.—Aged 35; married; one child; good habits; shoemaker. History on admission vague and trivial; said to have had a fever, and while convalescent from it, to have been frightened by a dog. On admission very feeble and restless, and greatly emaciated; voided his excreta involuntarily; tongue tremulous, and was jerked back involuntarily on protrusion. Eyes very prominent and pupils large; speech very slow and indistinct; a great part of the time he had to be fed; deglutition difficult; constant vigilantia. In a short time became paralyzed in both left extremities.

Had tonics and anodynes and extra diet, but all to no purpose, for he continued to emaciate; his pupils enlarged more and more; his eyeballs became more prominent and his speech and deglutition failed, and he died from coma.

Post mortem twenty-four hours after death. The head only was examined. Calvarium of normal thickness and consistence. On opening the dura mater a large quantity of serum, more or less tinged with blood, flowed out, and on elevating it, on the right side, lying between it and the arachnoid covering the pia mater, or rather within the arachnoid, we found a cyst of tolerably firm darkly bloodstained false membrane, covering the whole upper surface of this hemisphere. It was firmly adherent to the dura mater, and more or less on the other side, and it contained over an ounce of red fluid, besides a firm blood coagulum. The membranes, where they were in contact with the cyst, were somewhat discolored from the serum, but exhibited no further indications of inflammatory action. Underneath the cyst, the cortical portion of the brain was firmly compressed, and of a yellowish color, and somewhat softened. The remainder of the cortical portion of the brain was somewhat discolored yellow, and in several places slightly softened, though not to any great extent.

On section we found the thalami optici, corpora striata, corpus callosum and fornix so much softened that their respective outlines could not be distinctly traced. The tubercula quadrigemina, crura cerebri and pons varolii were likewise softened, and the cerebellum was intensely congested.

Dr. Pliny Earle records two arachnoid cysts of this character in *American Journal of Medical Science* for 1847; Calmiel reports several in his *Treatise on Inflammatory Diseases, &c.*, in 1859, and in the January number for 1865 of the *Journal of Mental Science*, London, we find two similar cases recorded. This is the first case of the kind that has ever fallen under our observation, and we regret that we have not a fuller history of its incipient stages and development prior to admission.

*Case Seventh.*

R. R., aged thirty-eight. Married. Three children. A blacksmith of strictly temperate habits. Insane for two months and a half. Admitted 1864. He was depressed, self-accusing, timid and fearful. Speech slow and stuttering, twitching at angles of the mouth. General erethism and large appetite. All the above symptoms gradually increased and he became filthy, helpless and destructive, and died May, 1865.

In this case all the usual somatic symptoms of paresis were well marked, but the mental phenomena were unusual. Gloom and depression, fear and anxiety, with a partial realization of his condition and a great dread of the future, were the most prominent mental symptoms.

Cadaveric inspection, twenty-four hours post mortem. Head only examined. Calvarium was rather thinner than normal. Dura mater was adherent on both sides of falx for several inches along apex. Both flexures of arachnoid were much discolored, and within the cavity we found eleven ounces of a pale straw colored fluid. The whole of the cortical portion of the brain was much softened, and also the white part, though not so much as the former. The puncta vasculosa could not be discerned. The ventricles contained a little more than their normal quantity of fluid. All the gray centers so much softened that their outlines could not be traced, and when moved all ran into a creamy mass. Cerebellum intensely congested and somewhat softened. Medulla oblongata and upper end of spinal cord much discolored and slightly softened. About three ounces fluid in theca vertebralis.

*Case Eighth.*

G. H.—; aged 34; single; boat builder; common education; not religious; was admitted November 27th, 1863. His father had been insane, and his mother was an epileptic. He had always been of fickle, rambling, unsteady character, always changing from one kind of work to another. For some time had been living with a woman of ill-fame, and had several times been treated for venereal.

His insanity is said only to have appeared eight weeks before admission, and was characterized by sleeplessness, restlessness, wild business schemes, impeded speech, large appetite and general muscular paresis. He retained the same general delusions up to the third stage of his disease, and then became melancholy and lachrymose. Thought himself rotten, dead, &c., and on this account refused to eat. He gradually failed, and died comatose, March 5th, 1865.

Inspection, thirty-four hours post mortem. Body rather fat, in good condition. Skull normal. Dura mater adherent along apex from anterior to

posterior fontanelle, and sideways from this line about two inches. Lower or inner surface yellowish, tinged with dark, deep red color, showing chronic inflammation with discoloration from effused fluid. Arachnoid, dark yellowish color. Pia mater same color, and very easily peeled off from brain proper. All the vessels running from brain into pia mater appeared cut. About half a pint of serum in sub-arachnoid space and in theca vertebralis. Glandulæ pacchioni very numerous.

Dissection. Centrum ovale, major and minor, normal, except that puncta vasculosa very small, almost absent. Each lateral ventricle contained about two drachms serum. Central gray commissure wanting. Choroid plexuses normal. Corpora striata and tenia semi-circulari normal. Optic thalamus somewhat softened. Fornix, hippocampi, crura cerebri and tubercula quadrigemina all softened. Pons Variolii softened below. This probably from having lain in fluid. Cerebellum and its crura normal. Fourth ventricle and medulla oblongata normal.

*Thorax.* Right lung had numerous adhesions all round upper lobe; left lung at apex and along external border. Both upper lobes and middle of right were excessively congested, looking very much like the second stage of pneumonia. *Heart.* Normal. *Abdomen.* Liver, stomach, kidneys, spleen and pancreas normal. At the fundus, about one inch from border on posterior side, and in mesial line, was found an ulcer, round, with rough, ragged edges, large enough to permit the insertion of a man's index finger. Mucous membrane of bladder much thickened and congested, and all around ulcer a series of corrugations like radii existed. Ulcer probably due to chronic inflammation, or may possibly have originated from a syphilitic chancre, as he has had venereal several times. Peritoneal cavity contained about three quarts of urine undergoing decomposition, dark in color, of very offensive smell.

In this case the question might arise whether general paresis itself was not the result of venereal.

I present, in conclusion, the views of Parchappe, Belhomme, Salomon, and Calmeil respecting the pathologico-anatomical conditions of the disease under discussion.

M. Parchappe.—“All the facts afforded by pathological anatomy agree in affirming the inflammatory nature of the characteristic lesion of the cortical substance of the brain in general paralysis. The special character of this lesion is to affect simultaneously both cerebral hemispheres, principally in the anterior and middle lobes, and to be associated nearly always with an inflamed condition of the meninges, frequently with inflammatory softening of the gray substance of the intra-cerebral ganglia, of the cerebellum, and of the medulla spinalis, with a granular condition of the ventricular walls, and with induration of the white substance; and finally, very frequently with atrophy of the convolutions.

“The softening of the cortical portion, and the changes of this substance or of the meninges, offer, in the first or acute stage, all the characters of an inflammatory condition; a rose, lilac, or even amaranth color of the cortical substance, hyperæmia, pointed injection, extravasations of blood in the cortical substance or in the membranes, adhesion of the pia mater to the surface of the convolutions, sometimes separation of the pia mater and

collection of a sanious liquid between it and the cortical substance. At a more advanced period, if the patient's life is prolonged, hyperæmia is no longer found. The softened cortical substance has a pale, dirty gray or yellowish tint. At this period of the disease are found atrophy of the convolutions, serous effusion in the anfractuositities, with thickening and opacity of the membranes.

"This rapid sketch of the principal characters which belong to the essential elements of general paralysis, appears to me to be an unanswerable proof of the necessity of referring it to a distinct nosological species. It is in fact a morbid entity, different from all others—a disease which is produced by causes which bring on over excitement of the brain, generally in men, and during the adult period of life; whose symptoms may be summed up in general and simultaneous lesion of the intelligence, the voluntary motions and sensibility, which has for its seat the cortical substance of the hemispheres, and for its constant anatomical character inflammatory softening of the cortical substance of both hemispheres, which, aggravated by cerebral congestions, causing every day a more marked impairment of motion, intelligence and sensibility, terminates fatally in an attack of congestion, or by cerebral marasmus.

"Though simple insanity may not be characterized by any constant change in the cerebral structure, nevertheless the alterations which are frequently found in the brains of the insane, and which, as some observers assert, are always found there, have the greatest analogy with the alterations which are met with in paralytic insanity. These are hyperæmia and thickening of the membranes, hyperæmia or decoloration of the cortical substance, induration of the white substance, atrophy of the convolutions, and collections of serum in the anfractuositities of the convolutions. Besides, it is essential that the importance of appreciable organic lesions should not be overrated. Because no constant structural change is found in the cortical substance in simple insanity, which is therefore classed with the *neuroses*, and considered a purely functional disorder, shall we therefore conclude that morbid action can be set up without structural change in the organ; but functional passes into structural disease in the lowest grades of dementia by atrophy of the convolutions. In my opinion, simple insanity, from being purely functional disorder, becomes organic in those cases in which it becomes complicated with general paralysis."

M. Belhomme.—"I reported in detail fifteen cases, which proved that general paralysis depends upon the alterations which I am about to enumerate. Thickening of the membranes and their adherence to the cortical substance of the brain, which is removed with them; the different layers of the cortical substance are softened, and present various shades of color, red, yellow and brown. The central portions diseased are the medullary substance, which is strongly injected, of a yellowish tint, softened in different degrees, sometimes only to a limited extent, and very often one of the hemispheres more altered than its fellow. The ventricles, often distended with serum, the arachnoid lining their walls is often thickened, and the medullary matter in contact with it either harder or softer than natural. The central parts constituting the cerebral peduncle and the commissure are often altered, the septum lucidum destroyed, the fornix softened to a

greater or less extent, the corpora striata atrophied or changed in color, the optic thalami, forming the principal wall of the third ventricle, are more or less softened. The cerebral peduncles are less consistent than in the normal condition; the annular protuberance sometimes partakes of the general condition of hardening or softening; in fine, the fourth ventricle and the rachidian bulb present various degrees of unequivocal hardening or softening, and the cerebellum partakes sometimes of the general diseased condition.

"I conclude by expressing the belief that general paralysis is an encephalitis of a particular kind, an inflammation which is developed under the congestive form, a disorganizing hyperæmia which is established slowly, producing at first induration and afterwards softening of the cerebral substance. At the same time there is a gradual impairment of all the functions of the brain, motion, sensibility and intelligence."

M. Salomon.—"I shall include the description of the pathological anatomy of the disease under four divisions, each being referable to a corresponding symptomatic stage:

"1. *Leptomeningitis chronica* (16) (=the stage of mental alteration)

"2. *Periencephalitis chronica diffusa* (=the stage of mental alienation)

"3. *Degeneratio substantiæ corticalis cerebri* (or marasmus substantiæ corticalis= the stage of dementia.)

"4. *Atrophia vera substantiæ corticalis cerebri* (=the stage of amentia.)

"That the disease commences with leptomeningitis of a chronic nature is proved by the fact that in the cases where the patient dies in the stage of mental alienation, signs of a still persistent or recently terminated inflammatory process are met with in the pia mater (=lepto-periencephalitis.\*) If the patient dies in the third or in the beginning of the fourth stage, we constantly observe a change in the pia mater, the result of a preceding leptomeningitis. The process indicated under 2, is recognizable by the increased volume ("*trübe Schwellung*") of the cortical substance of the brain. The degeneration referred to under 3 has been demonstrated by Rokitsky. The atrophy mentioned under 4 is discoverable principally by the circumstance that when the patient has lived to the commencement of the fourth stage, the most superficial portion of the cortical substance, corresponding to the lamina nervea in the healthy condition, is changed into cicatricial tissue, giving the sensation, on feeling with the point of the finger the now nearly obliterated surface of the convolutions, of a firm brain, and of a certain fluctuation of the subjacent dissolved cortical portion.

"The honor of having demonstrated the anatomical changes in paralysis with mental alienation belongs to the Vienna school (Wedl, Rokitsky)

"K. Wedl † has, in every case of general paresis, demonstrated an hypertrophy of connective tissue in the small arteries and veins in the pia mater and cortical portion of the brain. On the outer wall of the vessel is a hyaline, imperfect layer of connective tissue studded with partly scat-

\* A contraction of lepto-meningo-periencephalitis.

† "Beiträge zur Pathologie der Blutgefäße." Wein, 1859.

tered, partly grouped oblong or rounded nuclei. This layer of connective tissue, projecting over a greater or less extent of the vessel, undergoes, with the nuclei occurring in it, in the direction from without inwards (from the periphery of the vessel towards its center) a fibrillar change. The veins of capillary structure can not resist the pressure, but are also drawn into this process, and are completely obliterated, and changed to corresponding bundles of fibers. The abnormal layer of connective tissue not unfrequently serves as a seat of deposit for finely divided olein and amorphous calcareous salts, while in other places calcareous depositions take place in the inner elastic and muscular layer. The small and slender cerebral vessels thus calcified can, on section, be observed in the cortical substance as a number of needle points. Wedl endeavors to explain the adhesion of the superficial layer of the cortical substance to the pia mater by the penetration of the grouped nuclei in the adventitious membrane of the pia mater to a certain depth into the cortical substance. When the pia mater is separated, a layer of the softened cortical substance often accompanies it, corresponding to the depth to which the nuclei have penetrated.

"The complete obliteration of the calibre of the small veins caused by this degenerative process, demonstrated by Wedl, must give rise to a considerable obstruction to the circulation, both in the pia mater, and subsequently in the cortical substance of the brain, with consequent ischæmia;\* to stasis, pressure, irritation and inflammation. All this produces a progressive aggravation of the cerebral symptoms, and disturbs the nutrition of the cortical substance.

"Rokitansky† has, in all genuine cases of paresifying insanity, demonstrated a considerable increase of the connective tissue enveloping the cortical elements. The pathologically augmented connective tissue is at first of a tough and viscid nature, and imparts to the cortical substance a somewhat looser consistence than exists in the normal state. The connec-



Colloid (and amyloid) metamorphosis of the cortical substance in the person affected with paresifying insanity. The pia mater is represented as separated with loss of a portion of the cortical substance. In the cortical substance, the superior white filamentous layer (lamina nervea) is replaced by a layer of colloid corpuscles of various sizes; under this separate colloid granules lie in a mass studded with numerous granular nuclei. Lower down are ganglionic cells swollen or changed to colloid bodies.

\* Virchow—ιδίω—to check.

† "Ueber Bindegewebeswucherung im Nervensysteme." Wein, 1857.

tive tissue subsequently, in the course of the disease, assumes a harder and more fibrous form. This excessive formation of connective tissue causes the breaking up of the nerve-tubes. Those are first attacked which constitute the lamina nervea covering the cortical substance of the brain; afterwards those which horizontally traverse the same and separate the several layers of cortical substance; lastly, the degeneration attacks also the nerve-tubes, passing singly through the grey substance. The nerve-tubes broken up by the pathological process, are changed into colloid or amyloid granules (granular cells, granular bodies), which are met with in variable quantity in the extending connective tissue. The ganglionic cells of the cortical substance are often found dissolved, and in a state of colloid degeneration. See woodcut on preceding page (after Rokitansky).

"The cortical substance has split asunder, and (in the third stage) yields to the least touch. In the transition to the fourth stage, the superior layer (corresponding to the lamina nervea in the healthy state) is in a firm and tough condition. The inferior layers still retain their pappy and soft state. The convolutions are now nearly obliterated, and the mass of the cortical substance is diminished in volume.

"In consequence of this pathological process, set in action by ischæmia, determination of blood, hyperæmia, or inflammation, the grey cerebral cells become destroyed, and changed to an inert mass.

"The constant changes met with in every well-marked case of fully developed *insania paresans*, are:

"1. In the *arachnoid*, results of previous inflammation in the form of condensation, diminished transparency, &c.

"2. In the *pia mater*, results of previous inflammation appearing as opacity and condensation of the vascular membrane.

"3. In the *cortical substance*, the consistence is looser than is normally the case. It is often pappy and soft.\*

"In addition we frequently have—

"4. In the *dura mater*, results of previous pachymeningitis exhibiting themselves in adhesion of the membrane to the inside of the calvarium, thickening, &c.

"5. In the calvarium thickening and hyperæmia.

"6. In the *sack of the arachnoid*, effusion of variable nature.

"7. *Pia mater* often intimately connected with the cortical substance.

"8. In the *ventricles*, more or less, abundant serous effusion. If the changes enumerated under 1, 2 and 3 are not met with, the patient has had some other disease than *insania paresans*.

M. Calmeil. "1st. The progress of general chronic inflammation of the brain and its membranes (*péricéphalite chronique diffuse*), is frequently marked by developments of an alarming character.

"2d. These developments are especially characterized by sudden manifestations of apoplectic, of comatose and convulsive symptoms, and by strong indications of paralysis, showing a marked tendency to localize itself in some one of the members.

\* When the patient has died in the beginning of the fourth stage, the cortical substance may appear resistant, and normal to the touch. The most superficial layer must in that case be removed, before the dissolved state of the subjacent tissue can be observed (17).

"3d. The breaking out of these phenomena is owing to sudden congestive and inflammatory *recrudescences* (sudden augmentations of the congestive and inflammatory condition.)

"4th. The progress of chronic phlegmasia is marked then by genuine accessions of acute encéphalite (inflammation of the brain.)

"5th. During the violence of these *recrudescences* (augmentation), the inflammation may *localize* itself in the cavities of the cerebral arachnoid, in the depth of one hemisphere of the brain, of the cerebellum—in the annular protuberance, the spinal marrow, and penetrates more deeply than usual into a certain number of places in the superficial grey matter.

"6th. The material alterations which may occur during these species of inflammatory attacks, can with difficulty be diagnosed during the life of the patient.

"7th. These alterations are, notwithstanding, easily understood.

"8th. They are indicated by the products of the sanguineous extravasations, by the fibrinous coagulations, by the cellular or granular deposits, by heightened vascular coloring, by variations occurring in the degree of consistency of the encephalic nervous element.

"9th. The sanguineous extravasations of the arachnoidal cavities, the inflammatory crust (*les couennes*), often found therein, the cystic sacks, which may form these also, arising from the congested or inflamed capillaries (*des capillaires fluximnes*), filled with globules of blood, undergoing for the time being the inflammatory influence (*l'influence phlegmasique*.)

"10th. It is the same with the sanguineous suffusions so often noticed, in the cases under discussion, beneath the visceral fold of the arachnoid. (*du feuillet viscéral de l'arachnoïde*.)

"11th. As to the granular deposits also frequently observed, whether they exist in the midst of the arachnoidal cysts, or in the substance (*trame*) of the pia mater, or in the centers which inflammation has produced in a marked manner upon certain places of the encéphalic mass, they have arisen in virtue of that kind of law of construction which impels them to self-formation, as soon as the living fibrine has been able to spread itself on the outside of the vascular ducts which everywhere carry and distribute the blood necessary for the nutrition of our organs and tissues.

"12th. The intercurrent phenomena of périencéphalite (*la périencéphalite chronique diffuse*) are soon followed in certain cases by a fatal termination.

"13th. More frequently, however, immediate death does not ensue, because the congestive state of the encephalic capillaries is soon partially dissipated; but many of these capillaries nevertheless continue to retain blood in excess, and the condition of the patients who survive these phenomena is much aggravated.

"14th. Finally, many of these patients do not die until they have suffered a great number of these incidental encephalic attacks (*d'attaques d'encéphalite incidentes*) and this circumstance explains the multiplicity and the gravity of those material variations which we sometimes find in our anatomical descriptions.

"15th. We should put under contribution all the resources of hygiene, all the contrivances (*combinations*) of an active and enlightened foresight,



to prevent if possible inflammatory *recrudescences* (*augmentations*) of *périencéphalite* (*la périencéphalite chimique diffuse*).

"16th. The antiphlogistic treatment is often necessary to attain this end, and cannot be resorted to with too great promptness and vigor when the unexpected development of comatose phenomena, or of epileptiform seizures, threaten the existence of the subjects attacked with *périencéphalite* (*la périencéphalite chimique diffuse*).

I append a table, showing the morbid appearances found in 122 cases, by Dr. Skae of Scotland:

Calvarium of unusual thickness .....	16
do do thinness .....	9
Diploe absent.....	7
Osseous projections from inner surface of cranium .....	3
Ossific deposits in dura mater.....	4
Tumors in dura mater.....	2
Abnormal adhesion of do. to calvarium .....	20
Opacity and thickening of arachnoid .....	82
Congestion of membranes.....	41
Abnormal adhesion of do. to convolutions.....	60
Arachnoid and sub-arachnoid effusion .....	76
Sub-arachnoid sero sanguinolent do.....	4
Sero sanguinolent do. into sac of arachnoid.....	4
Pus in do.....	1
Granular deposit in do .....	5
Coagulable lymph in do. at base of brain .....	6
Atrophy of optic nerves .....	4
Grey matter unusually pale.....	12
do do dark .....	27
do do of a veolaceous tint .....	11
do do softened .....	37
White matter softened.....	11
do hardened .....	21
Lateral ventricles contained serous effusions.....	50
do do do sero sanguinolent do.....	1
Granular deposit in living membranes of do.....	43

