to a realization of his true situation with absolutely no memory of how he got where he is or of what he has been doing since he started away from home. As the name indicates, this condition has been described as one of automatism, but a moment's consideration will serve to show that acts of such a complex character cannot be automatic acts. The fact that no recollection remains of what was done has been used to argue unconsciousness, but that is equally inconceivable. Hundreds of miles could not be travelled by an unconscious man without attracting attention. The mere fact that the patient has forgotten what occurred is no reason why he must necessarily have been unconscious. The defect is rather one of memory, except possibly in the very simplest cases, and the patient, though perfectly conscious of what he was doing at the time, has simply lost all recollection of it. The author has been able fully to demonstrate this condition in one case in which the period for which the patient was amnesic lasted three hours, and as a result he is convinced that the same condition might be found to exist in others. Some persons are especially liable to this form of mental disturbance, and it may repeat itself on the occasion of renewed intoxication. Its psychopathological basis is probably a dissociation of consciousness.

The *neuropathic state*, though the most pronounced cause of alcoholism, may be an effect. *Neurasthenia* and *hysteria* may both occur as the result of alcoholism; the former caused largely by the malnutrition and exhaustion following continued over-indulgences (*Erschöpfungsneurasthenie*), the latter often affecting the male sex. In these cases the alcohol probably only serves to bring to light latent hysteria. *Dipsomania*—a periodical impulse to drink—though usually an expression of inherited neuropathy, may probably appear as a functional derangement grafted on an hysterical diathesis as indicated by a recent case cited by Janet[18] of *hysterical dipsomania* (*dipsomanie de forme hystérique*). This periodical tendency is further manifested in those cases in which, after repeated attacks of delirium tremens, there continues to be a periodical return of delirium although the use of alcohol may have been long discontinued (*alcoholic periodic insanity*).[12] These patients grow progressively more demented.

Chronic alcoholism, whether interrupted or not by any of the forms of mental disturbance described in this article, tends to an ever-increasing dementia, *alcoholic dementia*. Mental enfeeblement is a symptom from the outset and is noticeable at first in the esthetic and moral sphere. The previously proud, well-dressed man becomes slovenly in his habits and unkempt in his appearance. Incapable of the close and continuous mental application of former years it becomes impossible for him to meet the requirements of his business or professional life and lying is resorted to in finding excuses. This is followed by moral obliquities of a more serious nature in which the sexual element is apt to predominate and result in medico-legal complications.[19] Memory is early and noticeably affected. The every-day affairs of life are forgotten, so that the subject of alcoholism neglects to keep appointments, forgets important business engagements, etc. Judgment and the reasoning faculties are similarly enfeebled, until finally the most profound degree of dementia is reached, hastened perhaps by apoplectic insults which are not uncommon.

Pathology of Alcoholism.—The gross pathology of alcoholism has already been indicated. Cirrhotic liver, chronic nephritis, fatty heart, chronic gastritis, arteriocapillary fibrosis, cerebral arteriosclerosis, cerebral hemorrhage, constituting in the ensemble a veritable *senium præcox*.

The microscopic changes in the brain have been studied by various investigators. Vas[20] found central chromatolysis in experimental alcoholism in rabbits. Andriezen[21] found chromatophilia, swelling of the cell bodies, and thickening of the nuclear network. Dehio[22] found chromatophilia. Berkley[12] has found by the Nissl method beginning chromatolysis and by the silver-phosphomolybdate method varicose swellings of the dendrites and disappearance of the gemmules.* Ewing[23] found extreme chromatolysis in spinal, medullary, and cortical stichochromes. In many cells the lesion had advanced beyond simple chromatolysis, the nuclei were eccentric, and the remains of the chromatic bodies appeared as fine granules.

As regards the other elements of the brain Berkley[12] has found degeneration in the walls of the blood-vessels with irregular bulgings; obliteration of the Virchow-Robin or intra-adventitial lymph space and partial closure of the His or extra-adventitial lymph space, thus interfering with the excretory output of the brain. Some of the capillaries were plugged with leucocytes. In many instances the perivenous spaces were so filled with polyneuclear leucocytes as to occlude the venous flow by outside pressure. In connection with the pathology of chronic alcoholism mention should be made of Deiters' cells (the scavenger cells of Bevan Lewis[15]), the distal prolongations of the lymphatic system of the cortex, which by their increased activity help rid the brain of the results of degeneration by excretion into the lymph circulation.

Treatment.—The treatment of *delirium tremens* and the other acute alcoholic psychoses should be supporting: liquid concentrated food predigested if necessary. The bowels should be kept free and the kidneys kept flushed by a goodly supply of fluid. Heart stimulants are often necessary, digitalis, caffeine, strychnine, to combat cardiac failure, and hypnotics to induce sleep and give rest. The latter should be carefully selected with reference to the patient's condition, depressing agents such as chloral giving place to safer ones as trional if there is much heart embarrassment. The after-treatment consists of abstinence from alcohol, tonics, nourishing food, and regulation of the emunctories.

The medicinal treatment of *chronic alcoholism* should be tonic and supporting. Strychnine for a general nervous and cardiac stimulant, capsicum and bitter tonics for the gastric condition and anorexia; attention to the emunctories, moderate exercise, baths, massage, and electricity for their general tonic effect; sedatives and hypnotics with caution, a modified "rest treatment" if there is marked neurasthenia, and later a sufficient amount of mental and bodily exercise to keep the patient healthfully occupied. In this connection serotherapy and hypnotism may be mentioned in passing. The former has given no definite results; the latter may be of some value in cases which exhibit a marked hysterical diathesis.

The matter of isolation is an important one. The author feels convinced that in all cases in which the habit is firmly fixed isolation is highly desirable if not imperative, as in these cases the patient is unable to resist temptation and, as soon as an opportunity presents itself, will lapse. After confinement for a few months, during which the patient is restored as far as possible to physical health, he is in condition to abstain if he wants to and is able; if he does not wish to or if he suffers from too great weakness of will, he will return to his old practices and his case is hopeless. If he does wish to stop drinking, however, he has been given the best possible opportunity, an opportunity which should be early extended in all cases and not offered when by long-continued indulgence the case is of necessity hopeless.

II. Opiumism.—*Causes.*—As in other varieties of narcomania the most important cause is the neuropathic diathesis. In this class of patients the habit is often initiated by the use of morphine to relieve the periodic pains of neuralgia, tabes, dysmenorrhœa, rheumatism, etc., or the mental depression incident to worry, loss of position, grief, and the like. A great many cases are unfortunately traced to the carelessness of physicians in prescribing the drug, and as if in retribution medical

* In this connection see article, by Weil and Frank, "On the Evidence of the Golgi Methods for the Theory of Neuron Retraction," Arch. of Neurol. and Psychopath., vol. iii., No. 3. The authors take the position that varicoseness of the dendrites and disappearance of gemmules as shown by the Golgi method are artefacts.

men furnish the largest quota of sufferers (fifteen per cent.).[19]

Symptoms and Diagnosis.—The symptoms of a single dose are at first those of mild stimulation of the mental faculties followed by a period of quiet, half-waking, half-sleeping, interrupted by multiform pleasant hallucinations (predominantly visual) which show no tendency to delusive elaboration in the waking state.[14] This condition is followed by malaise, headache, dry mouth, constipation, and nausea.

The physical and mental disorders resulting from long-continued use are well formulated by Peterson.[4]

Physical.—1. Anorexia and constipation (later diarrhœa often). 2. Cachectic anæmia. 3. Cardiac intermittence, and bradycardia. 4. Muscular weakness with tremor. 5. Miosis in the early stages, mydriasis later, with sluggish reaction of the pupils. 6. Impotence. Amenorrhœa in women. 7. The knee-jerks are often absent. 8. Diminished sensibility to touch and pain, and concentric limitation of the visual fields. 9. Headaches and localized shooting pains, neuralgias, and paræsthesias. 10. Sensation of feeling cold.

Psychical.—1. Simple elementary illusions and hallucinations, muscæ volitantes, tinnitus aurium. 2. Loss of will and æsthetic sense, irritability; moral perversion, as in alcoholic psychic degeneration, but with little failure of memory. 3. Diminished attention, incoherence of ideas, and easily fatigued intellectual powers.

The diagnosis can often not be made without the anamnestic data. The patients frequently deny their habit—mendacity is a prominent symptom, and they are often 'cute enough to find means of indulgence even though carefully watched. The moral degradation is pronounced and they will go any length to obtain their drug. Symptoms which should excite suspicion are periods of torpor and languor (*Schlaffheit*) in marked contrast to the activity of alcoholism, amounting at times to an inability to even sit up, occasional signs of stimulation, small pinpoint pupils, yellowish-brown cachectic complexion, and, above all, the numerous scars of hypodermic injections.

Psychic disturbances develop more often as the result of abstinence than of continued use. They may be characterized by predominating depressive (melancholia) or exalted (mania) effects, or a paranoid state may develop. As in other toxic psychoses there are apt to be present more or less confusion and a tendency to multiform hallucinations. Dementia is a rare sequel.[9]

Prognosis.—The prognosis is not good and except in such cases as are not complicated by neurotic or psychopathic taint or disorders relieved by opium, recovery is hardly to be expected.

Pathology.—Opium has much less tendency to produce tissue degenerations than alcohol and many persons continue for years to take small doses with no apparent harm. Ewing[23] has found in the autopsies diminution of the amount of chromatin, and a tendency to central chromatolysis (in alcohol the tendency is to peripheral chromatolysis[24]), minute subdivisions of the chromatic bodies and eccentricity of the nuclei.

Treatment.—The treatment of morphinism has to do with the removal of the drug and the symptoms of abstinence. Isolation is more necessary than in alcoholism as these patients make more effort to obtain their accustomed stimulant surreptitiously. It is well, in accordance with the suggestion of Dercum,[25] not to begin stopping the drug until the patient has been under treatment for a time, confidence established, and the general health raised to the best standard. The *ration de luxe* can then be rapidly withdrawn, in accordance with the method of Erlenmeyer,[26] leaving the patient on about 0.15 to 0.20 gm. morphine per diem, below which amount serious symptoms are apt to present themselves. From this point on the withdrawal should be gradual. Symptoms of abstinence, if they appear, are referable to the heart, stomach, bowels, and nervous system; they are circulatory failure, respiratory disturbance, pyrosis, vomiting, diarrhœa, tremor, general debility, an hallucinatory delirium, and sometimes profound collapse. Ball[27] has called attention to pollutions and erotomania which may result from abstinence. For the cardiac weakness digitalis or sparteine hypodermically should be used, for the pyrosis bicarbonate of soda; vomiting and diarrhœa should be treated in accordance with general principles (bismuth, etc.), opium being avoided. If the mental and physical symptoms become grave morphine should be given and will usually relieve them. The evening dose should be omitted last, to combat any tendency to insomnia, and full feeding, massage, and Turkish baths are valuable adjuncts.

Meco-narceine (Duquesnel's solution) has been used by Jennings[28] as a substitute for morphine for a few days only after entire discontinuance. It is unnecessary to call attention to the danger of cocaine for this purpose. Codeine has also sunk into disuse and the recently synthetized derivatives of morphine, heroin and dionin, cannot be said to be any better. Their use is founded on a wrong theory and is fraught with danger. Cases of serious addiction to codeine and heroin have been reported.[28]

III. Cocainism.—*Causes.*—Addiction to this drug has in a great many cases come about by attempting to substitute it for morphine, and as a result pure cases of cocainism were formerly more rare than at present. Cocaine has been used so much of late in dentistry, minor surgery, and especially nose and throat work, that knowledge of it has become more or less general. The victims are usually those who have commenced its use for its analgesic effects and are largely physicians.

Symptoms.—The symptoms resulting from the use of cocaine are those of marked stimulation. The pulse is increased, pupils are dilated. The patients are active and extremely talkative, often repeating their remarks a number of times; they are constantly busy (*Vielgeschäftigkeit*), some of them writing endless letters, and their whole appearance indicates an acute intoxication. The effects are, however, very fleeting and the dose has to be frequently renewed. Chronic addiction results in marked emaciation, cachectic anæmia, insomnia, sometimes epileptiform attacks,[29] and various paræsthesias, the most marked of which is a sensation of crawling under the skin ("cocaine bug"). In the psychic sphere occur incapacity for mental application, lessened moral sense, mendacity, irritability, impaired judgment, and sometimes the delusion of marital infidelity. These symptoms may be followed by mental confusion with hallucinations, but more characteristically by a paranoid state (*hallucinatorische Wahnsinn*). From true paranoia this is differentiated by the greater variety of delusions, those of paranoia being less variable, rather noticeable for their monotony.[14] In the paranoid state of alcoholism, on the other hand, the hallucinations are more stereotyped.

The abstinence symptoms are not so severe as with morphine and may not appear for several days.[30] Erlenmeyer[31] has called attention to a profoundly depressed, lachrymose, demoralized condition, with moaning and sighing which may supervene. The persecutory delirium may persist for a long time and constitute the patient a very dangerous individual.

Morphine and cocaine addiction may also produce a neuropsychopathic state with symptoms of cerebral neurasthenia—morbid impulses, insistent ideas, etc. The author has recently had such a case under observation, in which the patient suffered from a convulsive tic with mental depression and suicidal impulse. Recovery followed prolonged abstinence.

Treatment.—Isolation should be insisted upon. The drug may be withdrawn rapidly as the symptoms of abstinence are not marked as in morphine. The prognosis of deprivation is good, but relapses are pretty apt to occur.

IV. Miscellaneous Intoxicants.—Various other drugs may produce marked mental disturbances as a result of acute or chronic poisoning or habituation. The limits of this article permit only of their mention. They are chloral, cannabis indica, somnal, sulphonal, paraldehyde, ether, chloroform, antipyrin, phenacetin, trional, chloralamid, iodoform, belladonna, hyoscyamus, salicylic

acid, quinine, the preparations of lead, arsenic, and mercury, and the bromides.

The mental effects of poisoning from all of these is in the main an acute hallucinatory delirium with more or less confusion. If recovery is not complete a paranoid state may persist. *William A. White.*

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XII. INSANITY: GENERAL PARALYSIS OF THE INSANE.—General paralysis of the insane, also known as paretic dementia, dementia paralytica, and popularly as paresis (which term for convenience will be used largely in this article), is undoubtedly an old disorder, but its recognition as a distinct type dates back only to the early years of the nineteenth century. The occurrence of paralysis with insanity apart from that associated with gross organic disease of the brain had been noticed by earlier observers, but it is to Bayle (1822) and Calmeil (1826) that we owe the first definite recognition and description. Since then it has been, more than almost any other type of mental disorder, a special study of the alienist, and its literature has surpassed in extent that of any other. It has been called a disease of modern life on the presumption that only in comparatively recent times has it become prevalent, and there is much apparent reason in this presumption. Certainly only of late years has its prevalence been so notable, and its actual increase seems to be a fact that cannot well be contested, making all due allowances for defective diagnosis which undoubtedly affected the statistics to some extent up to within a comparatively recent period.

The definition of general paresis presents some difficulties. In the first place, it is a protean disorder and its symptoms have often in the past suggested to alienists that we have in it not one but possibly several distinct morbid entities. This, in fact, has been the contention of some of the French writers, and it is still maintained by some that the paralysis and the dementia indicate two distinct types. The pathology of the disease has also been in dispute, so that neither on clinical nor on pathologic grounds has there been up to a very recent date a basis for positive assertion of the unity of the disorder. The close relations, however, with tabes, and their apparent convertibility in some cases with the more recently acquired facts in regard to the pathologic processes in the neurons in the latter affection, have of late years thrown much light on paresis. Among the very latest investigations in this direction are those of Mott, who holds that paresis is a disorder primarily due to a degeneration of the neuron itself, a parenchymatous degeneration, with secondary inflammatory changes affecting the vessels, lymphatics, and membranes, due to the irritation of the products of nerve decay. Accepting this theory of the disorder, which seems, in view of all the facts, as possible as any, we may define paresis as a generally subacute inflammation of the brain, due primarily to toxic degeneration of the nerve elements, especially those of the higher cerebral centres (speech, ideation, etc.), and generally attended with a chronic progressive dementia and physical paresis with manifold other mental, sensory, and motor symptoms, and ending inevitably in death from general exhaustion or from one or other of the special cerebral complications incident to the affection—acute delirium, convulsions, apoplectic attacks, etc.—or from intercurrent complications. The fundamental conception of the disease is the special nerve-cell involvement as the essential lesion of paresis, the other pathologic conditions being secondary. It does not necessarily follow that other pathologic conditions may not simultaneously appear and coexist with the degeneration of the neuron, but they are not the disease itself.

ETIOLOGY.—*Predisposing Causes.*—The first, if not the only, place in the causation of general paresis must be given to syphilitic infection. It is true that many, while recognizing the importance of this factor, nevertheless deny that it is by any means universal. The earlier writers on the subject, as a rule, paid but little attention to luetic infection among the causes of the disease; it was only incidentally mentioned by Bayle, and Esquirol recognized it only as contributing indirectly through the overuse of mercurial treatment. Sandras, however, in 1852, called syphilis and intemperance the two great causes of general paralysis; and some years later several Danish physicians, Steenburg, Esmarch, Jessen, and Kjellberg, announced their belief in the specific nature of the disease. Most authorities, however, up to the last two decades have put syphilis in the background, and it is only within that period that the compilation of numerous statistics indicating its frequency as an antecedent has brought it prominently to the front. In 1886 Rieger deduced from such figures as were then available the causal relation of syphilis, showing that the liability to paresis of those infected was sixteen or seventeen times as great as that of non-syphilitics. Since then the evidence has become much stronger, and a much higher ratio could be easily deduced. In 1891 the writer, analyzing the data afforded by the histories of two hundred and thirty-four paretics in the Illinois Eastern Hospital for the Insane at Kankakee, Ill., found a sufficiently clear evidence of syphilis in seventy-two per cent. of those cases in which any data could be obtained, and strong indications or probability of it in seventeen per cent., leaving only eleven per cent. in which there were no positive facts or probability. This did not imply that syphilis could be positively excluded in any case, but simply meant that there was no other evidence pointing to it than the existence of the paresis. Hougberg's figures are almost identical; he analyzed the facts in regard to exactly one hundred paretics, all that were on the records of the Imperial Lappvik Asylum, at Helsingfors, Finland. Of this hundred paretics, seventy-four per cent. had a clear history of syphilis. Twelve more had a less definite record of venereal disease, "ulcus mollis," gonorrhœa, or had been treated in the venereal wards of a hospital. Of the remaining fourteen, eleven had been dead ten years or more, and facts as to this special point were not so accessible as in the more recent cases. Nevertheless, several of them had a record of what appeared to have been possibly antiluetic treatment with the iodides, and others were of the classes that furnished a large proportion of paretics or had had apoplectic attacks in early life or had lived irregularly. One of the remaining three was sixty-three years of age, which arouses a suspicion of organic senile changes rather than paresis, and the other two were as it were waifs, only a short time under observation and lacking history. Any or all of these may have been syphilitics, as Hougberg says; it could only be said that it was not

in the histories that could be obtained—they were simply non-committal on this point.

Hougberg's statistics have been often quoted, and they are especially valuable since he took particular care and apparently had special facilities in looking up the antecedents of his cases. They are amply supported by recent statistics, though there are still some investigators who appear to have trouble in finding syphilitic antecedents in their cases. Berkley, for example, finds only fifty per cent. of proven and doubtful syphilitics altogether, and some French authorities have reported ridiculously small figures, making syphilis much rarer among paretics than among the general population. The tendency of medical opinion, however, of late years is to make this disorder the chief if not the sole predisposing cause, and Krafft-Ebing's catch phrase of "syphilization and civilization" as the primal causes of paresis is taken seriously to a large extent. The proof of the importance of syphilis is not confined to statistics. There are other facts almost as strongly indicative, such as the rarity of the disorder where syphilis is unknown, and among classes that are least liable to it, and the converse—its frequency in cities and rarity in rural districts, the cases of juvenile paresis in hereditary syphilitics, and those of conjugal paresis where both husband and wife are alike affected. All these point strongly to syphilis as a necessary antecedent, though the evidence is not complete enough to say positively that it is universal. The lack, however, of evidence of syphilitic infection in a certain proportion of cases is less important when we consider the difficulties of obtaining information as to this in a very large proportion of undoubted cases of the disease. It is probable that those who have reported only a small percentage of syphilitics among their paretics did so on a comparatively limited information, and had they had the opportunity and the will to investigate more thoroughly their percentage would have been much larger. It is possible also that other toxic agents besides syphilis may occasionally produce the disease, or rather, we should say, prepare the way for it. While we are, perhaps, not fully prepared to say with Möbius, Sachs, and others that there is no paresis without prior lues, they are far more justified by the facts than are those who deny or disparage its agency.

Another factor in regard to which there are varying statistics and widely differing opinions is heredity. The reported frequency of hereditary insanity, neuroses, etc., ranges from thirty to seventy per cent., or even exceeds these limits. Some continental authorities, French and Italian, have attributed much importance to a cerebral congestive heredity, and Naecke considers that a certain cerebral predisposition, not very well defined by him, is one of the commonest and most essential conditions of its occurrence. Berkley seems to adopt this view, and considers this heredity as congenital predisposition one of the most important etiologic factors. The writer, in an analysis of over two hundred cases, found less than half of the amount of neurotic or vesanic heredity than was met with in ordinary cases of insanity, and this included search for evidence of the so-called congestive heredity as well as that of insanity or other neurotic predisposition. It is very probable that any cerebral weakness, such as that claimed to exist by Naecke, would predispose to the disease in conjunction with other etiologic factors, but I am doubtful as to its occurrence as a rule in paresis. Except in juvenile paresis, which is directly dependent upon a specific heredity, it seems to me most correct to attribute less importance to this element in this disease than in other forms of mental derangement.

Age has a certain obvious influence in the incidence of paresis. It is pre-eminently a disorder of the active period of life, most cases occurring between the ages of twenty-five and forty-five. This is the period of special stress upon the nervous centres, the time during which the brain is most taxed, and when overwork, anxiety, exposure, and dissipation are making their drafts upon the nerve centres under the stress of modern civilization. After fifty paresis is rare, and though cases are reported after sixty, the writer has never seen one that was clearly this disorder; certain cases of senile organic change in the brain cells often simulate paresis, and are probably responsible for some of the cases diagnosed in patients of advanced age. Juvenile paresis, occurring about the age of puberty or rarely earlier, is always the result of inherited syphilis.

Sex and occupation are also generally included among the predisposing causes of the disease. The great majority of paretics are males, but its increasing frequency among women is a common observation in more recent times. Women are less exposed to the exciting causes than men, and it is a noteworthy fact that in this country at least the majority of female paretics are married women, and a syphilitic history can sometimes be found and more often suspected in their married partners. Of the unmarried female paretics, many are prostitutes, who seem to be especially liable. Of three personally known to me, one was, I believe, of this class, and one of the others had had an illegitimate child.

Certain male occupations seem to favor the occurrence of paresis, and they are the ones, as a rule, that conduce to irregular habits, sexual, alcoholic, etc. Commercial travellers, railroad men, speculators, men about town, etc., furnish a large proportion of all cases, while clergymen, Quakers, farmers, etc., furnish few or none. It is a disease of cities rather than of the country, where, though morals may not be always better, syphilis is less prevalent and life is simpler and less intense. Race in itself has probably very little to do with the etiology of paresis, though much has been made of the apparent exemption of certain peoples in the arguments against the syphilitic origin of the disease. It is not only the predisposing causes that are requisite, but also the exciting causes, and if either are lacking the disease is not likely to appear. Syphilis is common among the Egyptians, but paresis is rare; but there is little in their uneventful lives to cause the strain on the nerve elements that is required, and the fatalism of the Mohammedan is also a safeguard. While it is believed by many specialists that the Jews are less frequently affected with syphilis than those of any other race and religion, Beadles finds them in London specially liable to paresis, which he attributes largely to their trading and speculative tendencies. Many of them, moreover, come to that city from sections of Europe where syphilis is very common and one might say endemic, so that they do not necessarily escape special risks of infection.

Reviewing the predisposing causes of paresis, the first place must be given to syphilis; it is a question, indeed, whether it is not an essential cause. Heredity can be regarded only as a secondary and non-essential cause, and the others—age, race, occupation, etc.—are of importance only as related to the other two.

Exciting Causes.—The exciting or immediate causes of paresis are numerous, but one stands pre-eminent; that is, mental overstrain and worry, including under these the stress and anxiety of business and the struggle for existence under conditions of modern civilization. The best proof of this is the fact that paresis is almost exclusively a disease of civilization and almost unknown among barbarians and populations living under simple conditions; it is a disease of cities, where the struggle for life is keenest, and rare in rural districts and away from the temptations and excitement that abound in the great centres. There are also direct evidences of the fact that mental strain is one of the chief exciting causes, if not the chiefest, in the personal history of paretics; anxiety, business worries, etc., are among the most commonly ascribed causes. As syphilis and other predisposing causes, however abundant, are apparently powerless to bring about paresis alone, some additional factor is required, and the last straw that breaks the back of the weakened nerve element in this disease is most often supplied by some mental strain, most commonly that of the class of which worry and anxiety are the types.

Alcoholic excess has been recognized as a cause from a very early date, and undoubtedly is an important fac-

tor in many cases. Its importance, however, has been exaggerated. Alone it seems to have been powerless in the past, when such excess was far more prevalent than in many regions where of late years paresis has made its greatest advances. In combination, however, with other factors, its influence is not to be underestimated, and it has been a precedent in a very large proportion of all the occurring cases. It is very natural to assume its action in producing the disease, since it is well known that a very similar though transitory clinical picture is sometimes produced by it alone. The chronic incurable paralysis with dementia of chronic alcoholism has in it also some suggestion of that of paresis, though the differences are wide enough to make the distinction between them easy.

Sexual excesses are another commonly attributed and no doubt an important cause. There is often a history of such excesses, but its significance is sometimes lessened by the fact that it is itself an early symptom of the disorder. Unless the history of sexual debauchery goes back over a considerable period of time, as it often does, too much weight may easily be given it as a causal factor. Mott, one of the latest writers on the subject and one whose work is in many respects the best as well as the most modern, considers this element in the etiology very important; excesses of this kind, he holds, may deprive the brain cell of what is directly essential for its nutrition. By itself, however, it is ineffective. Lechery of the most abandoned and abominable type has always existed among men, and in past ages even more than in the present, but paresis is essentially a modern disease.

It is a question sometimes disputed, at least by inference, whether syphilis may be a direct exciting as well as a predisposing cause. Cases with the clinical picture of paresis occurring after recent syphilis or in connection with its secondary or tertiary manifestations are classed by some as syphilitic pseudo-paralysis, a term introduced by Fournier but practically discarded by him in 1894. It is customary with many writers to point out diagnostic features of this type as distinguished from true paresis, some of which at least are certainly fallacious, and none according to the writer's experience absolutely reliable. It does not appear, however, to him that there is any impossibility or improbability of the genuine disorder appearing after recent luetic infection, unless we can show the impossibility or unlikelihood of the special disorder of nutrition of the nerve elements which we consider the essential lesion occurring except after a lapse of years. Cases do occur with every symptom characteristic down to the resistance to treatment and the fatal outcome in connection with comparatively recent syphilis, and the most natural course is to recognize them as true paretic cases. It seems therefore correct to include syphilis with other commonly recognized exciting causes, as trauma and sunstroke, but there is generally back of these last some other well-marked factor, and usually, if the facts can be ascertained, a luetic history.

The etiology of paresis or general paralysis is one of the most important subjects in connection with the disease; hence the space here given to it. The disease is practically incurable and fatal. Prophylaxis is the only real defence, and this depends upon its etiology. Summing up what we know and the legitimate deductions from the same, I think it can be said that while there is a possibility in exceptional cases of some other toxic agency exciting it, in by far the greatest number, and practically in all, it has syphilis as its essential antecedent. In most cases this seems to act by preparing the way, creating a condition of susceptibility to other active causes, usually after the lapse of years. These later factors may be various, but among them and probably cooperating in nearly every case is the element of mental stress, either as overwork, worry, or the excitement of the demands of modern civilization generally acting on the weakened nervous organization. Heredity, except as hereditary syphilis in juvenile paresis, probably plays only a subordinate part—less, indeed, than in other forms of mental disease. We cannot altogether neglect, however, the possibility of all the factors together working on the heredity of the race and producing a class of more easy victims of the disease. There may be something in Naecke's supposition of a congenital brain weakness tending specially in this direction.

Symptomatology.—It has been already remarked that paresis is a protean disorder. Its beginnings are insidious; as Regis says: "There is perhaps no disease that begins more gradually than general paralysis. Except when it begins with a congestive ictus its invasion is so gradual and insensible that it is almost always impossible to fix its real commencement, and its origin is lost, so to speak, in the darkness of the past." It is unusual to find the earlier manifestations reported in the histories, and at best it is the rule to obtain only vague impressions or indistinct recollections of facts. Sometimes, as intimated in the above quotation, the first thing noticed is a maniacal, convulsive, or apoplectiform attack; but it is probable that in some of these cases there was a gradual deterioration preceding the insult, which passed unnoticed even by friends and intimates.

It is customary in text-books to describe three stages of typical paresis: the preliminary or incubatory stage, the stage of active bodily and mental symptoms, and that of final paralysis and dementia. To a certain extent this division is justified, but the cases of abnormal course in which these stages are not clearly defined are so numerous as almost to invalidate the rule. In a very large proportion, however, there is a period in which the disorder is only partially developed though it has a wide range in its symptoms. As a rule the very earliest beginnings, as already said, are little noticed even by the patient himself, and sometimes the fully developed stage of the disorder seems to break out without any prodromal symptoms whatever. The first symptoms are perhaps as often as not indistinguishable to the observer from those of neurasthenia. The patient feels himself somewhat incapacitated, his memory is less acute, his capacity for mental exertion is diminished, he complains of nervousness, sleep may be unsatisfactory, he may complain of obscure neuralgic or what he takes for rheumatic pains, headache is not uncommon, and most authorities mention it as occurring especially in the frontal and vertical regions. Vertigo is often complained of. The system often seems to the individual generally out of sorts, there may be dyspeptic symptoms and constipation, or other irregularities. At first the incapacity is noticed only under stress of work, but after a longer or shorter time it becomes so constant and prominent that he may give up work or have to abandon it. In some cases there are spinal symptoms resembling those of locomotor ataxia, girdle sensation, and occasionally actual ataxia and pupillary symptoms or other ocular paralyses appear at this stage, to disappear later or to become less conspicuous with the advance of the cerebral disorder. Berkley specially cautions practitioners as to their diagnosis of neurasthenia in a patient in middle life who presents reflex pupillary disturbances. Anisocoria, which in the developing or developed stage of the disorder is a frequent pupillary symptom, may appear in normal individuals, so that its special significance by itself is not so great as that of some others. An absence of the light reflex, the so-called spastic myosis, is far more significant but is not so prominent to the casual observer. The pupils are small, the reaction to light is absent or sluggish, and this symptom may be one of the earliest of all to appear. The alienist or even the physician in general practice so rarely has the opportunity to observe the earliest precursory phenomena that it is well to bear this symptom in mind. Other facial signs noticed in the beginning are occasionally a peculiar lack of expression in the features, hard to describe but quickly recognized by an experienced eye. There seems to be already a partial paralysis of the muscles of expression, not analyzable into defects of special muscles, but rather uniformly involving all. This becomes marked in the later stages of paresis, but sometimes may be detected early. The reflexes, tendon and cutaneous, are apt to be exaggerated

in this early stage, at least in the purely cerebral phase of the disease, but this is in accordance with the neurasthenic type prevailing. A fine tremor may also exist in this preliminary stage, though it is not often remarked till later, when it becomes a prominent feature.

The earlier mental symptoms of the prodromal period are also of the neurasthenic type—inability to perform mental labor, lack of power of sustained attention, defects of memory noticeable in little things, and in recollection of recent events, etc. The intellect may be apparently clear on most things and the patient be conscious of his slips of memory and other defects, and consider himself only a victim of nervous prostration. There is marked increase of irritability as a rule at this time, and, what is of some significance, a degree of moral defect is occasionally very evident, sometimes showing itself in a neglect of the minor proprieties of life, but often also in an apparent loss of moral inhibitions in higher matters. The tastes may change, and the individual, from being refined and fastidious, may take up coarse indulgences, become careless in habits and gross in his appetite, vulgar in speech, and sometimes show a decided sexual depravity. The altruistic emotions, family affections, etc., may be noticeably impaired or lost, and a disgusting egotism be manifested in many ways. The upright honorable man of business may become utterly unreliable and tricky, and the sedate father of a family indulge in undignified and open immoralities—all this before there have been enough other symptoms of intellectual or physical derangement to cause their friends to suspect their real condition. This is, however, not always the case; the moral lapses may be slight, and only the gradually advancing general weakness or neurasthenic symptoms call attention to their disorder. Sometimes only the minute but important physical defects in manual dexterity are the chief ones noted; we have seen a stenographer affected with incipient paresis in whom, aside from a decided emotional depression, an inability to take notes accurately and quickly from dictation was almost the only noticeable symptom of this entire stage.

The emotional depression mentioned above is a common feature in many cases, and may be taken for a sort of neurasthenic melancholia, the depression masking the other symptoms. Sometimes the mental disturbance is hypochondriacal in type, and this may extend well into the later stages after the physical symptoms are well advanced. There is every variation in the degree of intellectual impairment; in some exceptional cases it may seem to be slight even late in the disorder, so that a general paralysis without dementia has been spoken of by some writers. As an almost universal rule, however, mental defect to a greater or less extent is detectable on careful observation even in the earlier stage, and is the characteristic feature throughout the disease. It shows itself in the fatuous business projects, the confusion of ideas, the lapses of memory and propriety. It is also seen frequently in the writings, which sometimes show characteristic traits of mental failure, not only in their substance but in dropping of words, or letters, incomplete sentences, etc. They may also give evidence of the physical breakdown in a fine tremor which is characteristic even in this early stage, as already mentioned.

The Developed Disease, First Stage.—The transition into the second stage (first of some authors) is usually gradual and the distinction between the two not always marked. We may say that a good landmark showing the full onset of paresis is to be found as much as anywhere in the alteration of speech. When this becomes noticeably or even slightly embarrassed in the pronunciation of words demanding the use of the labials and dentals, the condition is recognizable by any one with the slightest experience. It is not only the difficulty of producing certain articulations, but a general defect of co-ordination of speech muscles that causes a peculiar thickness of utterance noticeable in any attempt at articulation. The patient will have special difficulties with some sounds, words like "National Intelligencer," "Peter Piper picked a peck of pickled peppers," etc.; he will stammer and fail and try to repeat, but the pronunciation of all words will be also affected. There is a defect of co-ordination of the muscles involved, together with a partial paresis, central in its origin. This symptom varies in extent in different patients; in some the speech alteration may be comparatively slight, but it is sufficiently frequent to be characteristic, and can be detected in some degree in nearly every case.

The muscles of expression are still more involved than in the prior stage, and irregular twitchings and local paralyses, lasting or temporary, are observed. There may be ptosis, one side of the face may be more affected than the other; tremors of all the facial muscles and of the limbs are characteristic of this stage. Manual dexterity is apt to be lost or very much impaired in special acts, such as those required in a trade or in the arts, all on account of the loss of co-ordination of the finer cerebral mechanisms. The muscular power in itself is not so much impaired as is the controlling power. The patient may feel well, all the bodily functions may be carried out, sleep, appetite, digestion, and excretions be normal or apparently so, for a considerable period after this disorder has thus fully developed; the reflexes differ little from those of the initial stage—that is, they are variable, the neurasthenic exaggeration may continue in the deep reflexes, or they may be diminished or lost. The pupils are unequal in a very large proportion of cases at this period, and may continue so to the end; in other cases there is myosis, such as is common in the earlier stage. In some cases the Argyll-Robertson reaction is marked. There is no regular febrile movement, the temperature may be about normal, except in connection with some accident of the disease, convulsion, or apoplectiform attack.

In a few cases ataxic symptoms precede the outbreak of paresis, as already mentioned, and they may occasionally appear in this earlier period of the fully developed disease. Their early occurrence and subsequent disappearance seem not to have been noticed by the earlier writers; the tabetic type has been generally recognized only of late years. Its comparative rarity accounts in part for this, and the fact that the tabetic symptoms are probably prominent only in the earlier ante-asylum stage is sufficient for the rest. It is, in fact, not of special importance, except as showing the relationship between the two disorders, paresis and tabes. Out of nearly two hundred and fifty cases under the writer's personal observation, definite tabetic symptoms occurred only in three, though it is possible the number might have been increased had full data as to the earlier manifestations been obtainable. In this form or stage—the ascending spinal type—the knee reflex is absent.

The congestive or apoplectiform and convulsive attacks which may occur, and which as previously stated may be sometimes the earliest manifestation, are most frequently seen after the paresis has fully developed. The apoplectic insults are less frequent than the convulsions, and, as a rule, more serious. Their onset is sometimes similar to an ordinary hemiplegic attack, though the unilateral paralysis is not always complete. There may be no loss of consciousness. A more characteristic paretic type is one in which the patient becomes suddenly or gradually comatose, with hot skin, stertorous respiration, and this leaves behind it a temporary paralysis of one or more members and generally a permanently more or less deteriorated mental condition. Or there may be only a temporary coma or somnolence of varying duration without very appreciable sequels; there is no regularity in the type or the results. I have seen a patient hemiplegic on one occasion and only aphasic on another, each time for the space of a few hours, but appreciably worse mentally after each attack. Occasionally these attacks are serious, and death may occur in the first seizure both of the congestive and the convulsive types.

The epileptiform attacks may occur singly or in rapid succession; the latter is a frequent type in the later stage of the disease. In either case they are often very similar in their appearance to those of epilepsy. They may,