need more light on this subject before we formulate any conclusions that are worthy of serious consideration.

DR. C. H. JOHNSTON—I do not say that the milk of every tuberculous cow shows tubercle bacilli, because this is not the case, but it has been proven repeatedly that the milk of tuberculous cows may contain tubercle bacilli, even though there is no demonstrable tubercular lesion of the udder. The detection of a tuberculous udder in its incipency is difficult even for an expert; hence, it may often happen that milk contains tubercle bacilli though no tubercular lesion can be demonstrated in the udder.

DR. I. A. ABT, Chicago—I wish to enter a protest against the idea that there are only a limited number of cases of tuberculosis which are congenital in origin. There is no doubt in my own mind that the number of cases of congenital tuberculosis which occurs in young infants must far exceed the origin in any other way. When we think that children, three months, six months or a year old often die of tuberculosis, although they have been under the best possible hygiene, it seems to me reasonable to suppose that these cases are of congenital origin. The only proof of the hereditary origin of the disease would be the demonstration of tubercle bacilli in the placenta or in the endometrium.

DR. EDWIN ROSENTHAL—Dr. W. P. Northrup presented a paper on "Tuberculosis in Children" before the Philadelphia Pediatric Society, and showed that the very inception of tuberculosis in children is through the lymph nodes primarily.

DR. NOER—I should like to know if any one present can produce a single case in which tuberculosis has been transmitted from the cow to a child. I am not aware that it has been demonstrated that bovine and human tuberculosis are the same.

DR. J. C. COOK, Chicago—I have under observation a lady sick with tuberculosis. It began as a cervical tuberculosis. A most minute inquiry as to the source of infection leaves very little doubt about its having arisen from a pet monkey. This animal had been sent away two years previously because it had tuberculosis.

DR. C. F. WAHRER, Fort Madison, Iowa—It would take a person with much fortitude to feed his children on the milk from a tuberculous cow. I wish to call attention to the specimens exhibited here by the Army Medical Museum. In this collection are several very beautiful ones of tubercular lesions of the udders. I advised a tuberculous mother not to nurse her infant, but to feed it on cow's milk. The cow presented every appearance of being healthy. The child died a few months later, and was found to be tuberculous, showing the importance of examining the animal before accepting the milk. Even if transmission of bovine tubercle to children can not be proved to take place directly, it were surely a good precaution not to feed a child milk from a tuberculous cow.

DR. E. F. BRUSH, Mount Vernon, N. Y.—Some years ago I had a cow suffering from acute miliary tuberculosis. I took the animal to another part of the farm and killed her. I shut up 24 laying hens and a rooster and fed them with tubercular masses from the lungs and intestine of this cow. I selected for this experiment hens because their body temperature is high, about 103 F. Some of them developed tuberculosis within eight days after beginning the feeding experiment. All of them died within five or six weeks from the time I began the experiment. I took the last 24 eggs laid by these hens and set them under healthy hens. All of these eggs developed a chick, but every one of them was dead. I took four of the dead chicks down to the Carnegie Laboratory, but through some mistake or carelessness, they were not examined nor reported upon. I purpose to repeat the experiment.

In regard to the transmission of tuberculosis from cattle to children, I would say that at one time I found a cow with acute miliary tuberculosis and killed her. There was a large tubercular encysted mass in the cow's mediastinal space, which she had probably had for years. The gentleman who owned this cow had brought up seven children on her milk, and although these children must have been fed on tuberculous milk, they remained healthy and did not develop tuberculosis. There must be something else besides the mere presence of tubercle

bacilli to start the tubercular disease. The additional element, I think, is an elevation of temperature. I think tuberculin is doing an immense amount of harm, and that its use is entirely unnecessary as a means of diagnosis. I know of a herd that is kept tuberculous by testing the animals with tuberculin. It is a very easy matter to make a diagnosis of tuberculosis in a cow when the disease has advanced far enough to do any harm.

DR. CLIFTON SCOTT, in reply—I wish to thank the members for this interesting discussion. I was much pleased to hear Dr. Abt point out what must be the conditions to have congenital tuberculosis. I do not believe that congenital tuberculosis is so frequent as Dr. Abt seemed to indicate, but the fact that a cow may be tuberculous and not give tuberculous milk unless there is a lesion of the udder points strongly to the fact that the mother may have tuberculosis and yet if the endometrium is not tuberculous during pregnancy there is not much probability of infecting her child. I have always felt a little guilty about not having discovered that the mother in my case was tuberculous before her confinement, for I might possibly have been able to save that child. I did not know that the mother was tuberculous, yet I think if I had been given the opportunity to give the case sufficient attention before confinement I should have discovered this fact.

THE ETIOLOGY OF PARETIC DEMENTIA.*

FRANK P. NORBURY, M.D.

JACKSONVILLE, ILL.

The lesion of paretic dementia is that of a diffuse parenchymatous atrophy of the nerve cells and part, at least, of the neuroglia. This process is essentially chronic and involves more or less the whole of the cortex, even extending to the basal ganglia and the cord. The well-known vascular changes start in the minute arterioles and in a slow but progressive manner involve the nutrition of the cells thereby causing degeneration of the neurons and neuroglia. The cortical substance undergoes atrophic changes and the lymph spaces dilate or widen; the lymph currents are interfered with—they are laden with debris—and ultimately the combined processes produce profound degenerative changes.

The question of the cause of this now well-recognized degeneration is seemingly in dispute, but I dare say that we all feel that there should be but little controversy over this question, when in the light of the accumulated knowledge of to-day we find syphilis paramount as an etiological factor. In fact, it is the one factor—the primary factor—and with Dana we can say "if there were no syphilis there would be no paretic dementia." Other authorities endorse this view, notably Mongeri,¹ who says "syphilis is necessary for the disease to develop; it is the factor which prepares the soil, while alcohol and heredity merely aid in its development." "Without syphilis, congenital or acquired, it is impossible to have general paralysis." These statements do not seem to lack verification when we consider the inoculation experiments, detailed by Krafft-Ebing, where in a series of cases, typical examples of paresis, in which by strict inquiry and examination the previous existence of syphilis could not be ascertained and in the face of this negative history, the patients were inoculated with virulent specific virus and yet after the allotted time for infection no evidences of syphilitic infection occurred. Not a sign or a symptom appeared, thus confirming at least by negative evidence that previous infection had occurred and serves to impress us

* Read at the Fifty-second Annual Meeting of the American Medical Association, in the Section on Nervous and Mental Diseases, and approved for publication by the Executive Committee: Drs. Frederick Peterson, Hugh T. Patrick and H. A. Tomlinson.

1. Am. Year-Book, 1901.

in the belief of the existence of latent syphilis in all cases of paretic dementia.

Paretic dementia certainly, then, has for a background syphilis. It is not in truth a syphilitic disease *per se*, but a para-syphilitic disease, due to the effects of the syphilitic poison. That is not a syphilitic disease, we know, by the therapeutic test. It is not amenable to anti-syphilitic treatment, in fact such treatment is utterly useless, although many practitioners persist in giving it a trial. The luetic poison of syphilis acts as do intoxicants—it is a toxin—there is auto-intoxication interference with nutrition and after its very early and prodromal stage causes a degenerative change of the nerve elements. There are no syphilitic lesions and in my experience the evidences of syphilis are, as a rule, seldom marked. In the beginning, the vascular stage of paresis, the lesions are those due to a toxin. There are none of the common lesions of syphilis such as noted in cerebral syphilis, viz., gummata and endarteritis. The toxin acts upon neural tissues to produce the morbid alterations, which, while at first they may be purely functional, ultimately become well-defined organic changes.

In the etiological study of about two hundred cases of paretic dementia, I found the history of syphilis in fully 60 per cent., which, in view of the fact that the history of many of these cases is meager, is yet conclusive as to syphilis in the general run of such cases. Inasmuch as it is impossible to confirm by inoculation tests the cases in dispute, we are disposed to believe that possibly many more of these cases are syphilitic and lack but the opportunity to establish the proof. I have sometimes thought that while we regard syphilis as the primary etiological factor, there may be other contributing factors, inasmuch as in my experience I have had several cases that, seemingly at least, have been pronounced or developed after some one of the infectious fevers. Thus suggesting the thought that these fevers assisted in some way in the development of the disease.

The following case, recently under my care, is an illustration:

A ranchman, with syphilitic history, who after successful years in business was induced to go to the Transvaal. Here he contracted pernicious malarial fever, from which he suffered greatly while in Pretoria. He was sent home and while en route it was noticed that he was acting queerly, developed mental peculiarities which in view of subsequent history were indicative of general paralysis. I say in view of subsequent history, simply to enforce what I said in a paper before the Illinois State Medical Society in 1892, that the early symptoms, the functional stage, of paretic dementia is not recognized from other forms of insanity by the attending physician and in consequence the prodromal stage is passed before the alienist meets the case. The stage when if any help is to be had for this disease it is to be given at this time. This patient developed into a typical case of paretic dementia and died after two years from the first appearance of the disease.

Another case first showed recognizable mental peculiarities during convalescence from typhoid fever, the mental symptoms being attributed to a post-febrile psychosis. This case was of the fulminating type, rapid in its course and terminated by death in three years. Another case with negative syphilitic history, during convalescence from an acute articular rheumatism, was noticed by his family to have certain marked changes in his disposition. They thought nothing of it after the family physician assured them "it would pass away."

But no, the case progressed, and rapidly too, resulting in death in about two years and a half after the attack of rheumatism.

I am sure that one swallow does not make a summer, but here are three cases coming under my observation, wherein after acute infectious fevers there has followed paretic dementia. Syphilis appears in the history of two—arthritic diathesis and rheumatism in one, all of whom had severe illness, just prior to appearance, at least of recognizable symptoms of paresis, and from such coincidences I am inclined to believe these diseases were contributing etiological factors.

The hereditary history of most of my cases shows that the neuropathic diathesis is the soil upon which this disease grows. Berkley says: "Heredity and syphilis are the most potent etiological factors." In his pathological findings he notes "microscopical evidences of irregular construction and anomalies in the cortical cells." From these facts we infer that there is a certain instability of the nervous elements which contributes to the susceptibility of an individual to the luetic poison of syphilis; that there is in fact selective action of this toxin upon neural tissue under certain conditions. This opens up that great question of immunity and susceptibility which is not to be discussed here. However, experience among the insane, including the cases of paretic dementia, leads all clinicians to form an opinion as to the family peculiarities, the hereditary considerations necessary in studying his cases, when he comes in contact with other members of the family than the patient himself. From this observation, however cursory, he draws the practical conclusion that heredity is certainly a factor and a potent one in paretic dementia.

Alcoholism is a contributing factor, but not of the importance ascribed to it by some. The brain lesions of the alcoholic are not those of paretic dementia and they simply are contributing degenerative conditions, which directly interfere with brain nutrition. I am told that in Colorado, alcoholism is usually a part of the history of such cases, but whether they are mere coincidences or not remains to be seen. I am inclined to believe alcoholism is coincidental in most all cases. I very much question cold, exposure and traumatism as being etiological factors; they can not cause such grave pathological changes. I conclude that:

1. Syphilis is the factor in chief which causes paretic dementia.
2. Infectious fevers with their toxic influences are contributing etiological factors.
3. Heredity is a potent factor.

DISCUSSION.

DR. HAROLD N. MOYER, Chicago—I do not know that I have any exception to take to some things stated by the essayist, but I certainly do not agree to all of the three deductions with which he closes his paper. I think we are not prepared to make the claim that paretic dementia is a para-syphilitic disease, nor do I think the author makes that claim. I know that in from 60 to 80 per cent. of cases of paretic dementia we get a syphilitic history, but does that justify us in saying that all the cases are syphilitic? If so, we must assume that because in a considerable proportion of cases syphilis is the etiological factor, the rest must have had latent syphilis which we were not able to recognize. I believe that such a doctrine is fraught with considerable error, because it is not justified by facts.

The hereditary influence in paretic dementia is very limited and is usually confined to cases of impure paretic dementia, those where we have it with a background of paranoia.

There is one question in relation to paretic dementia which is of considerable importance, namely, the racial distribution

of the disease. Among the natives of Hawaii, syphilis is a household disease; there is scarcely a native who has not been syphilized. Still, not a single case of parietic dementia has been recognized. There are other races among whom syphilis is very common, while parietic dementia is rare. The converse is also true. Recent statistics have shown that while syphilis is rare among the Hebrews in London, as compared with other Caucasians, parietic dementia is relatively quite common.

DR. E. G. CARPENTER, Columbus, Ohio—While my own experience is in accord with the general consensus of opinion that syphilis plays an important part as an etiological factor in paresis, still cases have come under my observation in which it was impossible to obtain any history or evidence of syphilitic lesions, and it is my belief that it is possible for paresis to be caused by factors other than syphilis. I believe that in the causation of many cases of parietic dementia, stress is an important factor. This, together with exhaustion from sexual excesses and the abuse of alcoholic stimulants, is sufficient to produce paresis in one having a neuropathic constitution. I think that in all cases we should be very cautious in asserting that a certain condition is due to syphilis, as there are circumstances where such a statement might do some people much harm; for example, in cases where the question of a pension and insurance is involved.

DR. RICHARD DEWEY, Wauwatosa, Wis.—I have seen at least six or eight cases of parietic dementia in which—as far as you can exclude anything—syphilis could be excluded, unless hereditary syphilis would be assumed. That assumption, however, can not be accepted without the existence of certain data upon which to base it. The statement made by Dr. Moyer, with reference to the racial distribution of syphilis and parietic dementia, leads me to refer to a confirmatory article bearing upon this subject which recently came under my observation. In this article the statement was made that while syphilis was very common in Mohammedan countries, parietic dementia was much rarer there than in non-Mohammedan countries, especially in Christian countries.

DR. SYDNEY KUH, Chicago—It seems to me that the question as to the etiology of parietic dementia, so far as anything definite can be said about it to-day, may be summed up as follows: That there is much evidence to show that syphilis is by far the most important etiological factor, both in paralytic dementia and in that other disease which is so closely allied to it, *tabes*. Whether or not syphilis is the only etiological factor it is impossible to say. The argument that in countries where syphilis is common, parietic dementia and *tabes* are very rare would prove nothing either in favor of or against the theory that there is some connection between the two conditions. The character of syphilis has changed completely within the last four centuries. When syphilis was first described, it had the character of an acute infectious disease which in a relatively short period terminated fatally. It is very possible that in certain countries the character of syphilis has assumed a different form from that which it has assumed with us. It seems that certain types of syphilis are particularly prone to produce grave degenerative changes in the nervous system of certain individuals. Instances have been recorded where two or three men were infected with syphilis by the same woman at practically the same time, and all developed the symptoms of *tabes*.

DR. EDWARD E. MAYER, Pittsburg—While it is a well-known fact that most cases of parietic dementia give a syphilitic history, it seems to me that we must place the relationship between these two diseases upon a more scientific basis. Because syphilis has occurred in a case of parietic dementia, it does not indicate to me that syphilis is the etiological factor. We should try to ascertain if the syphilis is responsible for the lesions. As the essayist has pointed out, the lesions found in parietic dementia are not those of syphilis. The time that has elapsed between the syphilitic infection and the onset of the parietic dementia should also be borne in mind. We should not at once jump to the conclusion that syphilis is the cause of the parietic dementia. That syphilitic toxins—whatever they are—may evoke nutritional disease of the brain-cells can not be doubted, sufficient, indeed, to produce pronounced disease, but

we must satisfy ourselves as to what conditions are produced by it, how it impairs nutrition and what action its toxins have. There may be other factors—the various toxemias, or exhaustion, or even traumatism may be the factor in the disease. I am willing to admit that syphilis is a most important factor in the causation of parietic dementia, but it should not be necessarily regarded as the only cause.

DR. H. A. TOMLINSON (chairman)—In considering the possible etiological relationship between syphilis and parietic dementia we should not lose sight of the fact that syphilis is a widely prevalent disease and almost as old as history, while general paresis, or parietic dementia, as the writer calls it, is comparatively infrequent, and, so far as we know, limited to the centers of modern civilization. Furthermore, when parietic dementia occurs in a syphilitic, the manifestations of the latter disease are not conspicuous and do not resemble other syphilitic affections of the nervous system, either in symptoms of somatic involvement or in its histology. Besides, syphilis frequently attacks the brain and produces degenerative disease, accompanied by dementia; that is not general paresis. It may also be claimed that the victims of syphilis and general paresis come from the same general class, and would therefore be equally liable to other possible causes of general paresis.

DR. FRANK P. NORBURY, in reply—I do not know that I can add anything further to the discussion. The fact is the Chairman covered the points that I had in mind. One of the speakers referring to stress as a factor in the production of parietic dementia, said that it was a prominent cause. In my experience this is not such a factor as is supposed, for comparatively few of my cases have come from the large cities—the busy centers of civilization—most of them come from the rural districts, and I am quite sure that they were not subjected to any special stress, either in connection with their occupation or social conditions. I believe the neurasthenic class alone succumb to stress. I have never seen a neurasthenic develop parietic dementia. In most of my cases the parietic dementia developed from fourteen to eighteen years after syphilitic infection, while several of them gave no history of infection at all. Yet from my knowledge of them as individuals I knew that they had often been exposed to such infection. In conclusion, I wish to emphasize the importance of the early recognition of parietic dementia. At the beginning of this disease there is a functional stage, as pointed out by Folsom some time ago, and if the symptoms are recognized during this stage we may be able to do something to arrest its progress. If allowed to go on, organic changes occur, with resulting dementia and death.

Clinical Report.

CASE OF SIMPLE FRACTURE OF THE VAULT, WITH MENINGITIS ON THE THIRD DAY. AUTOPSY.

EDWARD T. ALFORD, M.D.

INTERNE IN THE CHICAGO BAPTIST HOSPITAL,
CHICAGO.

The patient, Mr. W., while riding on a bicycle, Tuesday morning, June 1, was struck by a street car and severely injured. He was immediately taken to the Baptist Hospital, entering the service of Dr. Bayard Holmes, at 10:15 a. m. At this time he was conscious; there was no deviation of the eyes, no ptosis and the pupils were neither contracted nor dilated, and they both reacted to light. His hearing was apparently normal and there was no hemorrhage from the ears. There was no aphasia, as speech was normal, and no paralysis in any part of the body was evident. There was a good deal of hemorrhage from the nose and mouth, the upper lip being cut from the left nostril through the lower border of the nasal septum and through the lip a little to the left of the median line. This involved all the structures of the lip. A second wound on the inner surface of the mouth was found extending backward from the right angle of the mouth for