nineteen were done through a split rectus muscle and six through the oblique. The length of the incisions was from 1½ to 4 inches. Three had stitch abscesses; one was drained. I have included no operations done later than a year ago.

I wish here to express my indebtedness to a number of surgeons for kindly answering inquiries germane to this paper.

THE ETIOLOGY OF LOCOMOTOR ATAXIA.*

By J. O. HIRSCHFELDER. M. D., San Francisco.

OF ALL the nervous diseases, one of the most frequent and most interesting is tabes dorsalis. Its etiology, through the observations of Fournier and Erb, has been made so clear that few to-day will doubt its relation to syphilis. Large statistics, as well as careful investigations of individual observers, have proven conclusively that only a small percentage of cases is found in which previous syphilis cannot be demonstrated. When we bear in mind how often syphilitic infection is denied in cases of well-marked lues of the skin and other organs in which the cause is written upon the body in such plain letters that "he who runs may read," we will not be surprised that in a certain proportion of the cases of locomotor ataxia, lues cannot be proven.

In his latest pronouncement upon the subject, Erb has presented statistics of 1,100 cases observed by himself in which the question of previous syphilis has been most carefully considered. He finds lues to have preceded in 89½ per cent of the cases, whereas among 10,000 diseased individuals not affected with tabes, only 21.5 per cent of syphilis could be found.

His results are confirmed by observers in other lands:

Fournier in France found 93 per cent.
Collins of New York, 80 per cent.
Dana, 68 per cent.
Byron Bramwell in Edinburgh, 76 per cent.
Anfimow in Russia, 83 per cent.
Sarbo in Budapest, 86.6 per cent.

A collection of 6,000 cases collated from various authors gave over 80% of syphilis.

My very limited experience corresponds with these observations. Among 54 cases of locomotor ataxia observed at the Cooper Medical College Clinic, 35 had undoubted syphilis. With 5 the subject was not mentioned, whereas 2 were doubtful, and only 12 specifically denied previous lues. Among these, many, no doubt, either overlooked the previous infection or willfully prevaricated. However, leaving all of the doubtful cases entirely out of consideration, the 70% with whom syphilis had been observed speak loudly enough for the validity of the observations of others. It may be safely stated that nearly all unprejudiced clinicians of large experience to-day look upon syphilis as the only positive proven etiological factor in locomotor ataxia. A large number of other causes have been assigned to the disease, but the evidence upon which these assumptions have been made is not sufficiently great to bear critical scrutiny.

Dr. Collins, of New York, has presented his conclusions, drawn from a most careful analysis of 140 cases observed by him. He is unable to find any positive evidence pointing to any of the usually assigned causes of locomotor ataxia, such as sexual excesses, alcoholism, trauma, exposure to wet and cold, or worry, having any causal relation to the disease in the absence of syphilis. The acceptance of these deleterious influences as causes of tabes is merely another illustration of the tendency to receive statements without due and critical investigation.

However strong the clinicians may plead for the luetic origin of locomotor ataxia, there is great difficulty in persuading the pathologists of the truth of that doctrine. Virchow has always assumed an antagonistic position, and his teachings have been accepted by the large majority of pathologists. They are absolutely unable to find any indication of luetic origin in the far larger proportion of cases that come to autopsy. A small number of individuals who during life had shown the clinical symptoms of the disease, upon autopsy are found to have the gummous infiltration of the cord that is characteristic of syphilis, but these findings are quite rare. In the ordinary cases nothing can be found other than the usual sign of gray degeneration with increase of the neuroglia, such as will be found in any case of chronic myelitis.

Lately, however, Lesser has called attention to some facts which had been overlooked heretofore, and which may throw some light into this dark field. Lesser assumes that in addition to the secondary and tertiary forms of syphilis with which we are familiar, a quartan form may be recognized which manifests itself in interstitial infiltration in various organs attended with secondary parenchymatous degeneration. He finds these signs of quartan syphilis in most cadavers of luetic individuals, usually in the form of interstitial infiltrations in kidneys, livers and testicles. In these organs where the lesion is not extensive, the trouble is not recognized during life, as the function of the lost tissue is well compensated for by the healthy structure remaining. He calls especial attention to the syphilitic lobulated liver, to the syphilitic fibrous orchitis and to the smooth atrophy of the base of the tongue, the syphilitic character of which was pointed out by Virchow in 1860. In the latter disease we have an interstitial degeneration which is attended with a secondary degeneration of the glands at the root of the tongue.

In a similar manner Lesser believes that in the presumed quartan form of syphilis an interstitial myelitis develops with secondary degeneration of the axis cylinders of the cord, producing tabes. That the degeneration should be localized in the posterior columns seems no more strange to him than that the degeneration of the tongue should always be localized at the root only, or that the special form of orchitis fibrosa, which he considers quartan syphilis, should be restricted to the vicinity of the hilum. He likewise calls attention to the great frequency with which locomotor ataxia is associated with aneurism, a disease whose relation to syphilis is well recognized. Among 96 cases of tabes observed in the Moabit and Urban Hospitals of Berlin reported by Lesser, 18 were likewise affected with aneurism. Like all of these forms which Lesser considers quartan syphilis, tabes also appears many years after the primary affection, and shares with that disease the peculiarity of not being amenable to anti-syphilitic treatment.

A further confirmation of the syphilitic origin of tabes has lately been afforded by the cytological examination of the cerebro-spinal fluid removed by lumbar puncture. Attention was first called to this subject by Widal, who claims to have shown that in tabes and all other forms of syphilis of the cerebro-spinal tract, a marked increase of the mono-neuclear lymphocytes can be found. His observations have been quite generally confirmed.

It is unfortunate that we cannot, as a rule, prove the luetic character of tabes dorsalis by cure of the patient with anti-syphilitic remedies. It is true that a certain number of patients respond beautifully to that treatment. Everyone who has seen and treated many cases will occasionally have been gratified by the results, but in the larger number of instances neither mercury nor iodid will bring about any improvement. Yes, on the contrary, with many there is a distinct and well-marked increase of the trouble. This resistance to anti-syphilitics we have learned from Lesser is peculiar to all of the quartan forms of syphilis. The frequent occurrence of the paralyses of the oculomotor nerves and of the optic has always

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been a stumbling-block in the way of all explanations of the histogenesis of locomotor ataxia. The frequency with which oculomotor paralyses occur in syphilis not attended by tabes seems to confirm the luetic theory.

In 1894 Edinger, of Frankfort, proposed a theory on the etiology of nervous diseases, and especially of tabes, which should not be disregarded. This theory is based upon the fundamental proposition that the molecules of a cell are in a state of labile equilibrium such that if one part becomes enfeebled, it is overthrown and strangled by its neighbors. In a similar manner components of tissues are hypertrophied at the expense of enfeebled constituents. When a cell performs its functions normally, its contents are destroyed, but are immediately replaced in the regeneration which occurs. If the cell be overexerted or enfeebled by toxins or other poisons, the connective tissue luxuriates, and we have an interstitial hypertrophy resulting. There is no question that in the normal performance of function, substances are destroyed which must be replaced. The difference in histological appearance and in reaction to stains between cells that have performed their function and those which have rested has been clearly demonstrated by Daschkiewicz, Hodges and Nissl.

Edinger presupposes an increased vulnerability of the spinal cord through the influence of syphilis, possibly through a toxin. He calls attention to the fact that of all nerves of the body, those which rule over the maintenance of the equilibrium are the most frequently called into action, and are those which suffer the greatest wear and tear in the ordinary functions of life. It is just these organs of coordination which are injured the soonest and the most severely in locomotor ataxia. Edinger ascribes this fact to his theory of diminished recuperation. According to him, the first thing that occurs in the luetic individual who is about to become tabetic is a diminished recuperation of the system of nervous elements that bring about coordination. In consequence thereof, the uninjured neuroglia tissue luxuriates, and a sclerosis results.

In the spinal cord, when an injury occurs at any one point, secondary degeneration of the whole system to which that part belongs results, and as a consequence we have the anatomical changes characteristic of tabes. This explanation renders it clear why tabes nearly always begins in the lower extremities and arises so frequently in those who exert the lower extremities to a great extent.

A careful examination of the mode of life of those who become affected with high tabes arising first in the upper extremities may lead to its explanation by this theory.

In a similar manner, Edinger explains the peculiar action of the pupils in locomotor ataxia. The pupils are normally constantly undergoing changes due to varying degrees of light, and the nervous elements involved in the reflex act are subject to constant strain. Through the diminished recuperative power, he presupposes the disturbance that we recognize as the Argyle-Robinson pupil results. Of the muscles around the eyeball, the levator palpebræ, the abducens and the rectus internus are the ones most frequently called into action, and as would be demanded by Edinger's theory, these are the ones most frequently involved in tabes.

It is by no means improbable that the atrophy of the optic nerve may be explained in a similar manner, and it will be of interest to determine to what extent eye-strain forms its etiological factor in the cases of locomotor ataxia in which it occurs.

Sanitation of Railway Cars in Kentucky was instituted in the latter part of 1904, and after some little trouble from the railway companies, the State Board of Health succeeded in enforcing its regulations. The supervision extends to cars of all sorts.

ACUTE DELIRIUM.*

By H. E. SANDERSON, M. D., Stockton.

THE closeness with which this affection is allied to the distinctly somatic affections met with in general practice, and the shortness of its course with lethal outcome, render it of more than passing interest to the profession at large, and entitle it to your earnest consideration at this time. When you remember that in the cases I am about to describe the patients were sent to us from considerable distances by rail, and in some cases from general hospitals, to die within a few days after admission, necessity of bearing in mind the existence and leading characteristics of the disease will be evident.

Status and Etiology. The present status of acute delirium is a much disputed point amongst alienists. It was recognized by the ancients who termed it phrenitis. In 1884, Luther Bell described it under the name of typhomania, since which time it has also been commonly called "Bell's Disease." Spitzka, in 1883, first used the term delirium grave. Other writers have failed to mention it as a clinical entity until recent times, and even now much confusion is caused by trying to place all cases manifesting delirium in the same category.

Coming down to the present time, we find a great diversity of views amongst writers. Clouston speaks of it as a phase of severe primary mania, and calls it delirious mania. He terms it "a further stage of acute mania," and takes issue with other authors in their prognosis of the affection. Kraepelin calls it a "symptom complex" and not an independent disease. He views it as a pathological condition developing in the course of various psychoses—especially dementia paralytica, mania, acute alcoholism, and active melancholia. He terms it an irritative brain lesion of sudden development soon passing into paralysis. But, while maintaining that similar symptoms may follow in the wake of pneumonia, myocarditis, and septicemia, he admits that it may occur as a primary brain lesion. The majority of French psychiaters, de Boismont, Ball, Brand and others, look on acute delirium as being an individual disease. Mendel, Furstner, Meynert, Schuele and Alzheimer regard it as a symptom complex. Dr. Soukanhoff is of the opinion that acute idiopathic delirium is by its evolution and course nearer in semblance to an infectious disease than to anything else. The same opinion was held by Briand, Bianchi, Puncinnino, and Rosari. Berkeley thinks it a matter of doubt as to whether it should be considered an entity or only a symptom complex. He says "in the few cases it has been my fortune to see, some diagnosis other than delirium acutum—principally post febrile or meningitic delirium—would have been equally justifiable," and cites the fact that similar symptoms occur after a host of infectious troubles—especially pneumonia, typhoid, typhus, phthisis, dysentery, measles, acute rheumatism, influenza, the puerperal state, and gastric cancer.

Kellogg regards it as a distinct form of mental disease, and says the term should be limited to those independent acute delirious manias which present the typhoid symptoms and run the hyperacute course described. He speaks of suppurative kidney and pulmonary disease, and surgical operations accompanied by prolonged anesthesia as causative factors while some cases can only be accounted for on the theory of perverted metabolism and auto-toxemia. Krafft-Ebing considers it a distinct affection due to "certain injurious circumstances acting upon the brain, primarily through the vasomotor nervous system, causing hyperemia, through paralysis of the vessel walls." He mentions long-continued hard struggle for existence as a common factor, though in a large majority of cases the patients are predisposed to nervous troubles and excessive vasomotor irritability. Schuele

* Read before the San Joaquin County Medical Society, December, 1904.