ANGINA PECTORIS.

CLINICAL LECTURE DELIVERED AT THE POLYCLINIC HOSPITAL.

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GENTLEMEN,—Angina pectoris, or stenocardia, is a paroxysmal disease which is characterized by intense pain in the region of the heart, a sense of suffocation, a fear of impending death, and very frequently a slow pulse. The pain usually radiates from the chest into and down the left arm, although sometimes it extends down both arms. It is a disease which, like asthma, epilepsy, and migraine, has a tendency to manifest itself in the early hours of the morning, and after death, in many cases, there is found more or less degeneration of the cardiac muscle, of its arteries, and of its nerves.

Now, what is the intimate nature of angina pectoris? Has it a local habitation in the heart, or does it extend throughout the whole arterial circulation? Those who believe that it is a strictly cardiac affection pin their faith on the knowledge that the heart is the principal organ which bears any marked evidence of degeneration after death. This is in great part true, but it must be remembered that there are a number of so-called functional diseases which kill but leave no discoverable morbid traces behind. Our means of investigation are not yet accurate and fine enough to detect all the evidence which is wrought by affections of this sort. This statement pertains especially to diseases of the nervous system,—a class to which angina pectoris undoubtedly belongs.

Angina pectoris, then, being a recognized nervous disease, what are its essential features, and what portion of the nervous system does it invade? In answering this, I think the facts warrant me in saying that it is a disorder of the nerve-influence which dominates and regulates the tension of the arterial circulation; that this influence, as in epilepsy and in asthma, is thrown out of equilibrium and deprived of

its inhibitory power, and, in consequence, a spasmodic contraction of the heart and blood-vessels follows; and that the degeneration in the heart and blood-vessels is secondary to the perverted innervation.

What, now, are the facts which go to support this opinion of its nature? In the first place, Dr. Brunton has unmistakably shown, from a series of sphygmographic tracings, that the blood-pressure rises greatly during an attack of angina pectoris, or, in other words, that the arterial tension is largely increased during a paroxysm, and, in the second place, he showed that amyl nitrite relieves the disease by diminishing the blood-tension. Now, diminution of blood-pressure may be brought about in one of two ways: first, by lessening the power of the heart; and, second, by dilating the blood-vessels; and by a series of ingenious experiments Dr. Brunton demonstrated that amyl nitrite lowers the blood-pressure in animals, not so much by weakening the action of the heart as by dilating the large and small blood-vessels, and that in all probability this dilatation is produced through the influence of the vaso-motor nerves. The sphygmographic tracings and the physiological action of amyl nitrite concur in showing that the essential nature of angina pectoris consists in a spasm of the circulatory apparatus.

AORTIC REGURGITATION AS A CAUSE OF ANGINA PECTORIS.

Angina pectoris, then, being essentially a convulsion of the heart and large blood-vessels, it is quite clear, from our general knowledge of the etiology of disease, that any cause which lowers the nutritional tone of the nerves which supply the circulation will also become an exciting cause of angina pectoris. One such cause is aortic regurgitation, which, as we shall see, is capable of producing a failure of nutrition in the cardiac muscle and in the wall of the arteries, for the disease in question is infinitely more liable to occur in disease of the aortic valves than in disease of any other valves of the heart. Indeed, it is a question whether it is ever associated with affections of the mitral, tricuspid, or pulmonary valves alone. I collected from the literature of heart-diseases fifty-seven cases in which angina pectoris accompanied valvular lesions, and found the valves affected in manner and frequency as follows: aortic regurgitation, forty-four; aortic stenosis, two; aortic regurgitation and stenosis, four; aortic and mitral regurgitation, five; mitral disease alone, none. The significance of these proportions becomes still more manifest when we reflect that mitral disease is perhaps the most common of all the valvular lesions. Thus, of one hundred and ninety-two cases of valvular disease, casually collected, the mitral

was affected ninety-four times, the aortic forty-seven times, the aortic and mitral thirty-four times, the aortic and tricuspid once, and the mitral and tricuspid sixteen times, showing that mitral disease is nearly twice as frequent as disease of the aortic valve. This indicates very strongly, then, that exclusive disorder of the mitral, tricuspid, and pulmonic orifices and angina pectoris are, at least, not concomitant conditions, and that aortic disease is favorable to the generation of the latter disorder. Is this peculiar relationship only incidental, or is it a signboard which teaches us an important lesson concerning the pathology of the disease under consideration? I am not one who subscribes to the doctrine of chance, but believe that all things are governed by immutable law, and I am convinced that here is an instance of such government, if rightly interpreted.

Now, what relationship does aortic disease bear to angina pectoris? Does it stand as one of its causes, or as one of its effects? When we carefully consider the baneful influence, both direct and indirect, of aortic regurgitation on the heart and large blood-vessels, I do not see how it can require any stretching of the imagination to conceive a causal relation of this affection to angina pectoris; for during each contraction of the hypertrophied ventricle the blood is forcibly thrown into the large arterial trunks, distending these greatly and momentarily raising the blood-pressure to a maximum degree, while during diastole the blood rushes back through the leaking valve, empties the vessels almost completely, and suddenly drops the blood-pressure to a minimum point. This shuttlecock motion of the body of the blood not only exposes the blood-vessels to extreme oscillations of positive and negative pressure, but also weakens their resisting power and impairs their elasticity, and, in consequence, arteritis and other degenerative changes are set up in their walls. Much more than this, in health, when the valves are intact, the blood moves in a constant forward direction, and the heart and arteries are nourished by a blood-current the tension of which is comparatively constant. In aortic regurgitation, however, the blood is in a persistent to-and-fro movement, and the heart and blood-vessels are deprived of their requisite nutritive supply in consequence. In other words, these structures are bathed with blood, but suffer from poverty of the same, because it is too restless to be appropriated. It is obvious, therefore, why and how aortic regurgitation leads to greater degeneration in the heart and blood-vessels than can possibly occur in lesions of any other cardiac valve, and also why it may be regarded as one of the causes of angina pectoris.

URIC ACID AS A CAUSE OF ANGINA PECTORIS.

Angina pectoris is also a common affection among gouty and rheumatic people. This does not happen so much because gout and rheumatism, by producing endocarditis, have the special power to cause disorder of the aortic valves, and in this way, as we have seen, engender the disease under consideration, but because of the increased formation of uric acid, which is incidental to the gouty and rheumatic diathesis.

In *Brain* (Part I., 1891, p. 63) appears an article by Dr. Alexander Haig on "Uric Acid in Diseases of the Nervous System," which has a strong bearing on the subject under discussion, and from which, on account of its exceedingly great interest, I shall take the liberty of making liberal quotations. Being himself a sufferer from migraine, and knowing the close affinity between gout and this disease, Dr. Haig states that he was led to determine the amount of uric acid in the urine before, during, and after a headache, and always found that it preponderated largely during the attack. He also found that mineral acids diminished and alkalies increased the amount of uric acid excretion, and that acids relieved his headache and alkalies aggravated it. He furthermore lays down the proposition that, if other things are the same, "arterial tension varies with the amount of uric acid that is circulating in the blood." He says (p. 69), "I have accumulated many hundreds of pulse-traces which I think prove absolutely that the knowledge thus obtained gives me practically complete power over the rate and tension of the pulse, which in the great majority of cases is under the influence of uric acid in one way or the other. . . . On the one hand [p. 80], by diminishing the alkalinity of the blood, I can free it from uric acid, relax the arterioles, cause pricking and shooting pains in the joints, or greatly increase any previous gouty or rheumatic pains, quicken the pulse, and produce mental happiness and well-being, relieving headache or mental depression, if previously present. On the other hand, by increasing the alkalinity of the blood I can cause it to be more or less flooded with uric acid, can slow the pulse, contract the arterioles and raise the arterial tension, greatly diminish the excretion of urine and relieve the joint-pains, producing in their place, however, a sluggish circulation in the brain, with general languor, depression, and disinclination for exertion, and, if there is much uric acid, perhaps even headache or a fit." In other words, he holds that the presence of uric acid in the blood accounts not only for the sense of well-being and exhilaration before an attack of migraine or of epilepsy, but also

for the slowness of the pulse and the hardness and fulness of the blood-vessels during an attack of these diseases. That which is true of migraine and epilepsy in this respect is also true of angina pectoris, for it is an equally well recognized clinical fact that this disease is at least sometimes preceded by a similar feeling of exaltation, and is always, as we have seen, accompanied by a retarded pulse.

ONE REASON WHY ANGINA PECTORIS APPEARS IN THE EARLY MORNING HOURS.

Such, then, being some of the morbid effects of an excessive quantity of uric acid in the blood, the interesting question arises as to when the blood is most alkaline, and when, in consequence, the uric acid is most liable to preponderate in this fluid. We know that the blood is less alkaline when the body is doing active work than when it is resting, hence we have every reason to agree with Dr. Haig when he says that the acidity reaches its highest point about midnight, or in the small hours, and then gradually decreases until nine in the morning, when it is less than at any other time. From many pulse-tracings in his possession he is also able to show that the pulse-tension is highest in the early hours of the morning. "Such fluctuations in the excretion of uric acid and its amount in the blood occur every day, and it is only when they are exaggerated by errors in diet, by exposure to weather, or by the action of disease that their effects may be serious, and pass over into the region of pathology" (p. 79).

This, then, gives us a key to the problem why epilepsy, asthma, migraine, gout, and other nervous disorders are most apt to come on during the early hours of the morning. A periodicity at about this time seems to be characteristic of all functional nervous diseases, and this is precisely the time when angina pectoris is most prone to make its appearance, and, it being a spasmodic disease of the vaso-motor nerves, there is every reason to believe that uric acid is one of its exciting causes. Dr. Haig's paper is one of the most important contributions to scientific medicine which have been made for a long time, and it demonstrates, as its sequel proves, that a search for the causes of disease inside of the body is of infinitely greater value to the progress of our art than the researches which have been made in the interest of an objective pathology.

WHEN DO AORTIC LESIONS GENERATE ANGINA PECTORIS?

The danger of the supervention of angina pectoris in a given case of aortic insufficiency depends very much on the seriousness of the

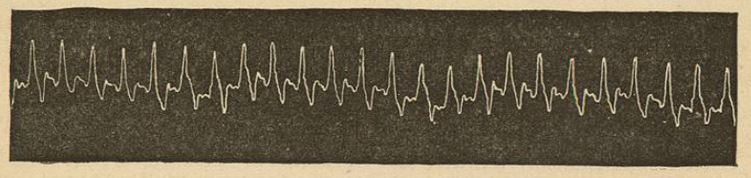
aortic lesion, and whether it owes its origin to a rheumatic, a gouty, or a nervous diathesis, as is shown by the histories of the two following cases, which are introduced here for the sake of illustration:

F., male, aged twenty-eight, had two attacks of rheumatism, the first one five years and the last one a year ago. Ever since the last attack he has been suffering from dyspnœa, especially on exertion, and also from a paroxysmal pain over his heart and chest, which comes on usually after midnight. The pain, which is also accompanied by a sense of thoracic constriction, is very severe, and radiates down both arms to the very ends of his fingers. During and for some time after the paroxysm his arms and hands feel numb and are partly paralyzed. Physical examination showed the existence of a double aortic lesion. His pulse beats eighty-four times in a minute. Under the use of rather large doses of strychnine, and of medium doses of phenacetin and guaiac, the attacks were checked entirely for two months, but recently have shown a tendency to recur, although in a more moderate degree than before he came under my care. The additional application of galvanism to the neck—the negative pole over the upper part of the sternum and the positive pole over the cervical portion of the spine—keeps the attack in abeyance. In this case there is obviously a rheumatic condition at the bottom of the trouble, which is as important from a therapeutic as it is from an etiological stand-point. The diathesis, which may be rheumatic, gouty, syphilitic, or nervous, often needs more consideration than the active disease itself. Remove this, and you frequently relieve the disease. This has been the principal aim in this case, and it has at least temporarily succeeded.

The influence of the absence of diathesis is well illustrated in the history of the case which is presented below, and which is almost a perfect fac-simile of the first case so far as the physical signs are concerned, yet there is no angina pectoris present. This is M., male, aged forty, who has been troubled with dyspnœa and an occasional fulness of the chest on exertion during the last six years. He is of powerful build, and has been accustomed to heavy work. He never had rheumatism, and follows the vocation of a market-dealer. Physical examination shows aortic regurgitation and stenosis. His pulse is seventy-two beats a minute. Now, both of these patients have double aortic lesions, —murmurs of about equal intensity in both, so far as the ear can tell, —yet their needs and general conditions are entirely dissimilar. The lesion in the former case originated through an attack of rheumatism, while in the latter it was brought about by overwork and lifting heavy weights.

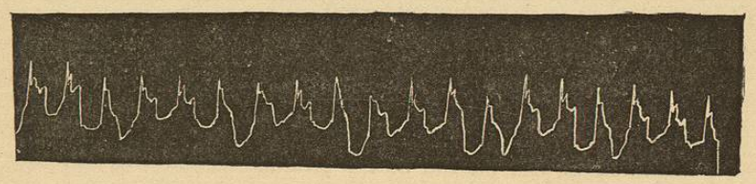
The accompanying heart-tracings, which have been taken from these cases, will in a measure assist in explaining the difference in the condition of the two patients. The first cardiogram belongs to F. and the second to M., both of them taken directly over the apex impulse of the heart.

FIG. 1.



Cardiogram belonging to F., to be read from left to right.

FIG. 2.



Cardiogram belonging to M., to be read from left to right.

In explanation, I would state that each pulse-tracing presents four points for consideration: 1st, the up-stroke, which represents the systole of the heart; 2d, the form of the apex; 3d, the down-stroke, which represents the diastole; and, 4th, the connecting curve between two succeeding impulses, which represents the auricular systole. (This is wanting in some beat-traces.) If, as in the tracing of F., there is an up-stroke which is almost perpendicular, it is an indication that the blood meets with very little resistance in being expelled from the left ventricle into the aorta, and that, in spite of the murmur which is heard during systole, there is very little actual stenosis present. The apex being sharp denotes that the heart empties itself fully into the aorta during each contraction. The down-stroke marks a very slight incline forward during its descent, showing that the aortic valves offer very little resistance to the back-flow of blood from the aorta into the ventricle,—the regurgitation being almost complete. The undulation in the line between the end of the lowest fall of the lever and the rise of the next indicates the auricular contraction. In the cardiogram taken from M., it is seen that there is a straight ascending line

and a sharp apex, indicating, as in the first case, no resistance to the outflow of the blood from the ventricle, although the murmur of stenosis exists. The line of descent is not perpendicular here, but is marked with jagged undulations, illustrating that the blood in regurgitating meets with greater obstacles than in the case of F.; or, in other words, the aortic valves are more patent in the case of M. than in that of F., and the regurgitation is not so large.

We cannot assume, however, that, because the patient M. has been free from angina pectoris up to the present time, he will always enjoy immunity from it. From what has already been said, it is very clear that aortic regurgitation has an innate tendency to bring on angina pectoris, no matter whether the aortic lesion arises traumatically, as from sudden strain (see page 31), or is of rheumatic or gouty origin: yet so far my experience teaches me that not all sufferers from aortic lesions die finally from this disease. Very frequently the mitral valves weaken and become leaky, and then such persons are cut off by intercurrent pulmonary disease before sufficient time has elapsed to produce disintegration in the way above indicated.

VAGUS DISEASE AS A CAUSE OF ANGINA PECTORIS.

Another cause of angina pectoris is found in disintegration of the pneumogastric nerves. From our knowledge of their distribution and physiology it is quite evident that disease of these nerves must necessarily manifest itself in disorder of the structure and function of the heart. Forty-five years ago it was shown by the Weber brothers that stimulation of the pneumogastric nerves with galvanism had the effect of slowing the action of the heart, and if the current was made sufficiently powerful the heart was arrested in diastole for a short time, after which it began to beat although the galvanization was continued. The influence of these nerves on the heart is also illustrated in the case of Czermak, who by pressing his vagus against a bony tumor in his neck could stop the beating of his own heart at will. Concato (*Virchow u. Hirsch's Jahresbericht*, 1870, Bd. i. S. 144) describes two cases similar to that of Czermak, in whom slowing of the heart could be produced by compressing the vagus on the right side of the neck. In both cases the position of the vagus and of the carotid was abnormal. The sense of thoracic constriction, with an urgency to take deep inspirations, etc., which Czermak observed in his own experience, was not present in these cases.

Furthermore, post-mortem investigation shows that when these

nerves are diseased they play an important *rôle* in the production of angina pectoris, as is attested by the following histories:

Case I. (Leroux, *Le Progrès Médical*, 1878, No. 27.)—Male, fifty-three years old, saddler, suffered from angina pectoris for seven years; the attacks came on from six to eight times a day, and the pain radiated into the left shoulder, arm, and lower jaw. After death, which came on suddenly, it was found that the left heart was hypertrophied, the coronary arteries open and normal, and both phrenic nerves and left vagus were healthy throughout. The right vagus was patent until it reached the right bronchus, to which it adhered, and was embedded in a peritracheal gland at the same spot. This gland was surrounded by hardened connective tissue, and was discolored on the surface and calcified in the interior. The vagus was involved in it for two centimetres, and at the place of compression there existed a thickening of its sheath.

Case II. (Romberg, "Diseases of the Nervous System," cited from Fothergill, "The Heart and its Diseases," p. 271.)—A man, thirty-six years old, complained of nervous symptoms, and of his heart standing still. It intermitted for five or six beats. The aspect of the patient showed that something terrible was going on within him; he sat as if thunderstruck, speechless, motionless, his eyes wide open, and his consciousness unimpaired. When asked about his sensations, he stated positively that for a second or often longer he had a presentiment of coming arrest in the shape of internal restlessness and oppression; that when the stoppage took place a violent pain seized both sides of the thorax, extending to the neck and then passing up to the nape and the head; that the pain remained fixed in the latter for some time after the attack; and that when the attacks were frequent he could scarcely get rid of the sense of weight at the cervix. The attacks were brought on by emotion. During the intervals the heart presented no abnormal action and was quite healthy. The attacks increased in severity, and he died in a state of torpor. After death it was found that the large cardiac nerve was woven into a black knot the size of a hazel-nut; the left vagus was involved in an underlying, nodulated, dark-blue lymphatic gland. The phrenic nerve was also embraced in the diseased gland.

Then, again, experimentation on animals makes it extremely probable that the pneumogastric nerves have a strong trophic influence on the heart-muscle, for Wasslief (*Virchow u. Hirsch's Jahresbericht*, 1881, Bd. ii. S. 139) concludes, from his experiments on rabbits, that the fatty degeneration of the heart-muscle is due to the elimination of the

vagus influence through vagotomy. He also shows that in starving pigeons fatty degeneration of the heart-muscle occurred much earlier in those in which the vagi were cut than in those in which they were not cut. In a research on the trophic relations of the vagi to the heart-muscle, Eichhorst (*Centralblatt f. d. Nervenheilkunde*, 1879, S. 111) used birds, dogs, and rabbits in his experiments, and concludes that a constant fatty degeneration of the muscle of the heart followed division of the vagi, provided the animals did not die too early of pneumonia.

There is reason for believing that the sympathetic nerves which largely form the cardiac plexus are likewise concerned in the causation of angina pectoris, for Lancereaux (*Gaz. Méd.*, 1864, p. 432) reports the case of a male, forty-five years old, after whose death from this disease it was found that a tumor had compressed several branches of the cardiac plexus of nerves.

EMOTIONS AND SUDDEN MENTAL SHOCKS AS EXCITING CAUSES OF ANGINA PECTORIS.

Taking into consideration the close anatomical and physiological relation between the brain and the heart, through the vagi on the one hand and the sympathetic on the other, we are in a position to comprehend how emotional influences and mental shock of any kind are capable of inciting a paroxysm of angina pectoris. In health, even, the heart is frequently disturbed by thinking of it, as is attested by the medical examination of applicants for life insurance. The fear that some possible lesion may be discovered by the examiner frequently accelerates the heart's action far beyond its accustomed limit and capacity. Romberg ("Nervous Diseases," p. 6) relates the case of Peter Frank, who, while concentrating his attention on the subject of heart-diseases, during the preparation of his lectures, was attacked with such severe palpitations and an intermittent pulse that he fully believed himself to be suffering from aneurism. All these symptoms disappeared, however, after his work was completed and after he had taken a long rest and relaxation. Tuke says ("Influence of Mind on Body," p. 87), "Some years ago a medical student in Paris, on being initiated into the mysterious rites of a Masonic society, was subjected to the sham operation of venesection. His eyes were bandaged, a ligature bound round his arm, and the usual preparations made to bleed him. When a pretence of opening the vein was made, a stream of water was spurted into a bowl, the sound of which resembled that of the flow of blood, which the student was anticipating. The consequence was that in a few moments he became pale, and before long fainted away. There

is a case on record of a man who was sentenced to be bled to death. He was blindfolded, the sham operation was performed, and water allowed to run down his arm in order to convey the impression of blood. Thinking he was about to die, he did actually die. Imagination had the same effect as the reality. But it is impossible to say how much fear had to do with it; probably a good deal, as in the instance of the man reprieved after his head had been laid on the block and the fatal axe was about to fall. The reprieve came too late. The anticipation of death had arrested the action of the heart."

John Hunter had an attack of gout when he was forty-one years old, and during the last fifteen years of his life he suffered and finally died from angina pectoris. He tells us that in his later life he became subject to violent disturbances of his heart when he was annoyed or anxious about any event. He says ("Works of John Hunter," edited by Mr. Palmer, 1838, vol. ii. p. 336), "At my country-box I have bees, which I am very fond of, and I was once anxious about their swarming, lest it should happen before I set off for town; this brought it on [an attack]. The cats tease me very much by destroying my tame pheasants, partridges, etc., and rooting up my plants. I saw a large cat sitting at the root of a tree, and was going into the house for a gun, when I became anxious lest she should get away before my return; this likewise brought on a spasm; other states, when my mind is much more affected, will not bring it on." Anger and anxiety had a very pernicious influence on his heart, and, knowing this, he said, "My life is at the mercy of any scoundrel who chooses to put me into a passion." And, indeed, his apprehensions were realized, for in a dispute concerning the admission of certain students to St. George's Hospital he was flatly contradicted by one of the governors. This so enraged him that he "immediately ceased speaking, retired from the table, and, struggling to suppress the tumult of his passion, hurried into the adjoining room, which he had scarcely reached when, with a deep groan, he fell lifeless into the arms of Dr. Robertson." An examination after death showed that his heart was small and strongly contracted. The coronary arteries had been changed into bony tubes, the aorta was dilated, and both the aortic and mitral valves were diseased.

SYPHILIS AS A CAUSE OF ANGINA PECTORIS.

The possibility that syphilis may produce angina pectoris is a subject of grave importance, and one to which sufficient attention has not as yet been directed. From our knowledge of the hydra-headed manifestations of this disease it would seem strange, however, if the heart escaped its contaminating influence. I have seen a number of cases of organic heart-disease and heart-pain the origin of which I attributed to a syphilitic source, but a true syphilitic angina pectoris I do not remember as having occurred in my experience. Other observers have been more fortunate in this respect, and during the last few years a number of such cases have been reported, and on account of their intrinsic value I shall abstract the histories of a few from an article by Professor G. A. Sacharjin, of Moscow, on "Die Lues des Herzens von der klinische Seite betrachtet" (*Deutsches Archiv für klinische Medicin*, 1890, Bd. xlvi. S. 388).

Male, aged forty, syphilitic, with frequent attacks of angina pectoris, which were followed by pulmonary œdema, and for which digitalis and other agents were employed to no purpose. Under an energetic course of sodium iodide treatment the attacks disappeared entirely, but returned later, although in so light a form that they were readily set aside by digitalis.

Male, aged fifty-five, infected with syphilis, although at an earlier period he passed through an attack of acute articular rheumatism, and was also addicted to the excessive use of alcohol. For more than a year he had suffered from severe attacks of angina pectoris, which were but indifferently relieved with nitro-glycerin. There was evidence of arterio sclerosis and aortic insufficiency, but aside from the stenocardiac paroxysms the patient was apparently well. Sodium iodide internally and inunctions of gray mercury ointment cured the attacks of angina pectoris, but had no effect on the aortic regurgitation or the chronic arteritis.

Male, aged —, is analogous to the last case. The attacks of angina pectoris were likewise entirely relieved by the same treatment.

Male, aged thirty-seven, addicted to alcoholic abuse for a number of years, and two years before he came under observation he acquired syphilis, which was followed by the customary secondary symptoms. Eight months previously he was suddenly attacked one night with angina pectoris. Two months ago he had his second paroxysm. Under the influence of sodium iodide and inunctions of mercury he was fully restored.

The author ascribes these attacks to the noxious action of syphilis on the heart and nervous system. What special portion of the nervous system he believes to have been affected is not intimated. May the vagi have been involved? That syphilis affects these nerves is attested by Vierordt, who relates a case (*Archiv f. Psychiatrie*, etc., Bd.