agreement ceases. In the one case, the further evolution of the brood is towards pus; in the other instance, the progeny become the formed elementary tissues of a cancer, a sarcoma, and the like.

Here seems to me to be the proper place to add a few words on the anatomy of *conical cornea*, since certainly, in its later stages, when the apex of the cone is nebulous, if not before, tissue-changes of an inflammatory or irritative type are present. The affection is commonly met with in young adults, who are generally of feeble constitution; and it usually affects both eyes, although often in unequal degrees. By the kindness of Mr. Bowman, I had, some years ago, an opportunity of submitting to minute examination a beautiful example of it. The conical part of the cornea was thinned. The attenuation increased from the base to the apex of the cone, the thickness of which was only one-third of that of the normally curved circumference of the cornea. The anterior and posterior epithelia and Dmours' membrane were unaltered; but the anterior elastic lamina was thinned, and the immediately underlying lamellated tissue was replaced by a stratum of crowded club-like nuclei. More deeply still, the corneal corpuscles were very numerous, dominating over the interstitial tissue, and actively multiplying. At the base of the cone, there was a gradual transition from the diseased to the healthy lamellated tissue; and the circumference of the cornea was perfectly normal.

[To be concluded.]

ON LOCOMOTOR ATAXY.*

By J. LOCKHART CLARKE, M.D., F.R.S., etc.

THE curious disease known by the name of locomotor ataxy is very imperfectly understood even by those who have had many opportunities of observing it. A great number of practitioners have never seen a case, or at least have not diagnosed it when they have seen one. It is not uncommon to find locomotor ataxy mistaken for some form of cerebral or spinal paralysis, and cerebral or spinal paralysis mistaken for locomotor ataxy. The pathological physiology of this disease is not well understood even by those who are more expert in its diagnosis; while specimens of its morbid anatomy have been seen only by a very few British practitioners, since I believe I am the only person in this country who has made it a subject of investigation.

For these reasons I thought that, while practitioners from all parts of the kingdom are now met together, it would not be uninteresting to select, and illustrate by specimens of morbid anatomy, two cases of locomotor ataxy, one of which presents all the symptoms characteristic of the disease, while the other is wanting in some of those symptoms, and therefore might be, to the inexperienced, a subject of difficult diagnosis. I shall not, however, now dwell on the differential diagnosis of the disease. Those who wish for further information on this point, I may refer to the writings of our distinguished visitor, Dr. Duchenne de Boulogne, whose name is identified with this disease, and to my own article on locomotor ataxy in the first volume of *St. George's Hospital Reports*. But, before I read the cases which I have selected, it may be well to give the briefest possible outline of the symptoms and course of the malady.

In a great number of instances, the first symptoms make their appearance in the form of strabismus, with diplopia, which may disappear for a time and then return; or in the form of amblyopia or weakness of sight, which may go on to complete amaurosis. After a variable period, these symptoms are accompanied by so-called "rheumatic" and lancinating pains, which occur at variable intervals, in different parts of the limbs. In many cases, the ocular disturbances, except perhaps extreme contraction of one or both pupils, never make their appearance; the pains, which may extend over months or even years, accompanied by some weakness, being the first in the train of symptoms. Either at the same time, or subsequently, there is commonly more or less numbness in the feet and legs, in the hands and arms, and sometimes in the face. Sooner or later, the patient begins to find that he cannot properly maintain his balance; that he totters in walking, like a man partially intoxicated, or that he cannot guide the movements of his fingers. He has lost, to a certain extent, the power of controlling the action of some of his voluntary muscles. Still later, the voluntary movements become more or less jerking or spasmodic; and in the course of the disease other symptoms supervene, as incontinence of urine and dysuria, which frequently alternate in the same patient; loss of control over the sphincter ani; generally, though not always, loss of sexual power and desire; occasional hypersthesia over certain

parts of the limbs; and sensations of tightness around the body and limbs. In rare cases, of which I have seen two, the senses of smell and taste are impaired. Usually, the patient's general health is not much affected, and his intellect remains unimpaired. Locomotor ataxy is a disease which occurs more frequently about the middle period of life, and is much more common in men than in women. This very concise outline must of course be considered only as a general introduction to the cases which I intend to relate.

MORBID ANATOMY.—The morbid anatomy of locomotor ataxy consists chiefly of a certain grey degeneration and disintegration of the posterior columns of the spinal cord, of the posterior roots of the spinal nerves, of the posterior grey substance or cornua, and sometimes of the cerebral nerves. A variable number, and frequently, in the latter stages of the disease, nearly all the nerve-fibres of the posterior columns and posterior roots fall into a state of granular disintegration, and ultimately disappear. Usually, the posterior columns retain their normal size and shape in consequence of hypertrophy of connective tissue, which replaces the lost fibres. In this tissue, at wide but variable intervals, lie imbedded the remaining nerve-fibres, with the debris of their neighbours, in different stages of disintegration. In some places they are severed into short portions, or into rows of globular masses, formed out of their medullary sheaths, or white substance, which has been stripped from their axis-cylinders. In other places they have fallen into smaller fragments and granules, which either lie aggregated in the line of the original fibres, or are scattered at irregular distances. Corpora amylacea are usually abundant, and oil-globules of different sizes are frequently interspersed among them, and collected into groups of variable shape and size around blood-vessels of the part. I am inclined to believe, from my own investigations, that, in the course of the disease, the posterior cornua of grey substance are *always* more or less affected, as I have elsewhere pointed out; and it appears to me to be a question whether they are not the first parts, or at least amongst the first parts, that are morbidly changed. I have also shown, and will show you to-day, that in some cases the deeper central parts of the grey substance are more or less injured by areas of disintegration. These latter lesions, however, are not essential to the production of locomotor ataxy, the peculiar symptoms of which depend solely on lesions of the posterior columns, of the posterior nerve-roots, and probably of the posterior cornua. The cases in which they occur may be considered as mixed cases, partaking of the nature of locomotor ataxy and common spinal paralysis, like the second of those which I shall describe.*

[To be continued.]

ADDITIONAL NOTES ON UNUSUALLY RAPID ACTION OF THE HEART.

By RICHARD PAYNE COTTON, M.D.,

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In the *BRITISH MEDICAL JOURNAL* of June 1st, 1867, I related a case, illustrated by sphygmographic drawings by Dr. Sanderson, in which the pulse reached 232 per minute. It was the first of the kind which had been published. In the *JOURNAL* of June 22nd of the same year is a letter addressed to me by Sir Thomas Watson, Bart., in which he describes a very similar case which had fallen under his notice several years previously, and where the pulse reached 216 in the minute. Dr. James Edmunds also described a like case (*JOURNAL*, June 15th, 1867). A short time afterwards, four other cases were recorded—one by Dr. J. D. Brown (*JOURNAL*, July 20th, 1867); two by Dr. R. L. Bowles (*JOURNAL*, July 20th, 1867); and one under the care of Dr. Broadbent at St. Mary's Hospital (*JOURNAL*, Aug. 3rd, 1867).

As this closes the number of cases hitherto placed on record, and the condition is, as Sir Thomas Watson justly remarked, "a very rare form of disorder," I have thought it might be worth while to add the following case, which has lately fallen under my observation.

A few months ago, I was requested by Mr. Langhorne, of 227, Brompton Road, to meet him in consultation upon a case of excessive palpitation of the heart. We found the patient—a gentleman aged about 35, and leading ordinarily a very active and anxious life—suffering with severe dyspnoea and general depression, accompanied by marked symptoms of gastric derangement and slight muscular rheumatism. It was impossible to count the pulse, the beats being far too quick, feeble, and apparently irregular; but, on placing the stethoscope upon the heart, we

* Read before the Medical Section at the Annual Meeting of the British Medical Association in Oxford, August 1866.

* All the morbid appearances above described were shown to members of the Association at Oxford, in preparations made by the author from spinal cords, etc., of cases of locomotor ataxy.