25. Tongue heavily coated, breath fetid, no appetite and very restless. Had slept but very little during the night. The discharge from the eye and sloughing areas was very profuse. At this time I prepared two slides, one from the fluid of a pustule and one from the pus discharging from the eye. Upon microscopical examination, both slides showed the short, thick rods of bacillus anthracis. In the evening of the same day I inoculated a tube of agar-agar, and after eighteen hours, examination showed the presence of bacillus of anthrax in large numbers, and upon this I based and made my diagnosis of anthrax.

October 23, edema increasing. Both eyes completely closed; discharge greater; pustules still forming, and could now be seen in all the stages, a very interesting condition to observe. The mucous surface of the left side of the mouth was covered with a grayish-white membrane resembling diphtheria and the surface would bleed freely when this was removed. Temperature 102.5; pulse 120; respirations 28, deep and labored. Very restless, some dyspnea and vertigo on rising. Could speak but little louder than whisper. Could not take any nourishment on account of swelling of mouth and throat. Photograph No. 1 shows condition of this day.

October 24, condition same as preceding day, except breathing was heavier and edema still increasing.

October 25, conditions same as before, except the swelling of the right eye was reduced sufficient to permit it to open slightly, but the edema had spread over the entire chest as low as the apex of the heart, and presented a bright red appearance resembling the rash of "iodism." Slight pain also developed in the bowels, which lasted for thirty-six hours. This was the only pain experienced during the course of the disease.

October 26, edema decreasing, discharge about same. Temperature 100; pulse 100, respirations 22. Voice better. Can take small amount of diet, and getting some natural sleep. Within the next twenty-four hours temperature went to normal, and never rose above 99, and improvement continued until patient was in good health.

On the tenth day a line of demarcation formed as shown in photograph No. 2. Within this line everything was gangrenous, except the eyeball and the deeper portion of the eyelids.

The sight of the eye was at no time affected, although the eyeball was highly congested. The gangrene destroyed all the soft tissue over the cheek, to the periosteum, and all the muscles about the eyes that control the lids.

Dr. Stuber, of Lima, Ohio, directed and treated the eye and has made three plastic operations, hoping to restore better functions of the eyelids, but the results have not been satisfactory.

Treatment.—Having no experience in any way with this affection and finding but little literature on the subject that seemed to meet the symptoms present, a line of symptomatic treatment was adopted, which consisted of quinin, whisky, diuretics and cathartics, internally and externally, local application of bichlorid cloths to all parts that were sloughing, boracic acid salve to the eye, after thoroughly cleansing all the discharge from the eye with hot boracic acid solution.

On the eighth day after inoculation I found an article by Vockresensy,⁴ in which he highly eulogizes the use of large doses of carbolic acid internally, claiming to have cured sixteen consecutive cases of malignant pustule, and in some it was not begun until the seventh day.

In my case it was not used until the eighth day; within twenty-four hours from the time of commencing it improvement was noticeable, and it was continued until all symptoms of sepsis had disappeared. Whether the favorable result was due to use of the carbolic acid, I do not know, but should I get another case I would begin it promptly.

The main points of interest are: 1. Does this one case prove that an anthrax center exists on this farm, the home of the horse and patient? 2. Did the horse really have an anthrax at the time he was suffering from the swelling and discharge from the head? 3. The difference in the symptoms and results of the two cases reported when the points of inoculation were so nearly the same. The virulency in either case proved to be sufficient to produce death in mice in twenty-four hours.

INTERESTING THROAT PARALYSES IN A CASE OF LOCOMOTOR ATAXIA OF AN IRREGULAR FORM.*

JOHN EDWIN RHODES, A.M., M.D.

CHICAGO.

Mr. F. D. R., aged 35, railroad employe, was sent to me Jan. 26, 1901. About four days previous to his visit he began to cough occasionally and had at that time noticed some difficulty in articulation, especially in the liquid sounds, *t* and *d*; the labials *m* and *n* were pronounced easily. At this time there was noticed occasional though slight regurgitation of liquids into the nasal cavity on swallowing. He gave a history of rheumatic pains in the shoulder some four or five years ago, but had had none recently.

He confessed to an attack of gonorrhea, but had never had syphilis, nor were there any general symptoms indicating such an attack. His habits were good; he did not use alcoholics, and used very little tobacco. His sense of hearing was good; the sense of smell was considerably impaired; vision in the right eye was poor.

There were no pains at this time from which he suffered, but he had had sharp lancinating pains in the lower extremities at one time for several years. His normal weight was 150 pounds, but now he weighed only 135 pounds. His strength was fairly good; the temperature and pulse were normal. There was no dyspnea, vertigo, or headache.

He complained of a slight tickling sensation in the larynx, which excited cough occasionally. The tongue was slightly coated, the appetite fair, the digestion good, but he was habitually constipated. Micturition was frequent and could not be controlled very well.

Dr. C. D. Wescott, who sent him to me, reported upon his case as follows: "In regard to the case of F. D. R., I would say that he was referred to us last May, by Dr. Patrick. He came with the diagnosis of locomotor ataxia, and was sent to us because of a drooping of the upper lid of the right eye and a divergence of the same eye. He was also complaining of pains in the eyes and head. He said that the eye first turned out two years before and had not been straight since. Upon examination, it was found that there was complete paralysis of the branches of the third nerve of the right side. The ophthalmoscope showed some opacities in the vitreous in the right eye and the retinal vessels were full and hazy in outline. The optic nerves appeared normal in both eyes. We prescribed glasses which gave him great relief in the use of his eyes for near work; and, about the middle of June, he reported himself decidedly better. There was noticeably less ptosis and slight movement of the right eye in was possible. We then lost sight of him, and he did not return until we sent him to you on January 26 complaining of his throat. On that day there was almost com-

4. Annual of the Universal Med. Sciences, 1891, vol. iii.

* Read before the Chicago Laryngological and Climatological Society, May 2, 1901. (See Proceedings, p. 1801.)

plete ptosis of the right upper lid, as well as divergence of the eye. His vision was not quite as good as it was at the time of our examination in May; the optic nerve was gray, possibly indicating beginning primary atrophy.

I asked Dr. Patrick as to his condition in May and he reported to me as follows: "I first saw F. D. R. on May 3, 1900, and found him suffering from rather atypical tabes. The right pupil was dilated and had been so for two years. Occasionally the left eye would swing outward, giving him double vision. He had well-marked incoördination, diminished knee jerks, the left more so than the right. I regret to say that my record makes no mention of the pupillary reactions, but I feel quite sure that they were not normal. I could get no history or other evidence of specific infection."

Dr. Gill had seen the patient previously and reported as follows: "F. D. R. called to see me about two years ago; he told me he had been suffering from sharp lancinating pains in the lower extremities for several years. An examination of his condition at that time showed exaggerated patellar tendon reflexes; ptosis of the right lid with sluggishness of both pupils to the light. There was no incoördination, and no ataxia. The last examination, which occurred several weeks ago, showed a change in some of the symptoms, inasmuch as the reflexes had disappeared entirely. The lancinating pains had persisted, but still there was no ataxia or incoördination. He has difficulty in urinating, an inability to completely empty the bladder, also inability to control the sphincter ani at all times. There were some areas of anesthesia about the chest. His trouble is probably an irregular form of locomotor ataxia."

I found the nasal cavities comparatively normal. When the mouth was opened, and the tongue depressed for an examination of the throat the soft palate was in a normal position. On irritating it with a probe, however, the right side of the palate was contracted strongly to the pharyngeal wall, drawing the uvula toward the right, the left side of the palate not responding at all to the irritation, but remaining relaxed and stationary. There was no anesthesia of the parts.

On examination of the larynx the right vocal cord was stationary in the median line, there being a paralysis of the posterior crico-arytenoid on the right side—the abductor of the vocal cord. All other conditions of the throat were normal.

The diagnosis then was, an ascending sclerosis of a locomotor ataxia causing ptosis of the right eyelid, and divergent squint of the right eye, paralysis of the left half of the palate, and abductor paralysis of the right vocal cord.

The motor nerves affected were interesting. The palate derives its motor stimulus from the otic ganglion of the fifth, supplying the tensor palati; the facial supplying the levator palati and azygos uvulae. The recurrent laryngeal supplies the posterior crico-arytenoid; the third supplies the levator palpebrae, and the sixth, the external rectus of the eye.

The diagnosis should not be difficult. The throat conditions might be confounded with those found in the acute form of bulbar paralysis, but the rapidly progressive nature of this disease with its symptoms would enable one to easily exclude it. In this case the history, with the symptoms and signs of ataxia, although not altogether typical, satisfactorily settled the diagnosis as due to a progressive and upward sclerosis involving finally these nerves at their origin in the bulb.

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THE DIAGNOSIS OF DIAPHRAGMATIC HERNIA.*

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The diagnosis of diaphragmatic hernia presents many difficulties in the living subject; so great indeed are these that the condition is seldom recognized until operation or death has yielded an ocular demonstration. The affection is found in rare cases at autopsies and it is occasionally discovered in the dissecting room in bodies dead of other affections, but all told only a little over 300 cases have been reported. H. I. Bowditch reported a case in 1847 and at that time collected reports of 88 cases. Leichtenstern¹ reported a case in 1874 and at that time collected reports of 250, but only five of these were diagnosed before death. Thoma² collected 290 cases in 1882 and many more have since been reported, but still the obstacles to diagnosis remain, although as shown by Leichtenstern, and by Abel³ in 1894, there are some cases in which a reasonably positive diagnosis may be made without great difficulty. As I have elsewhere stated⁴: "This affection possesses many symptoms and signs in common with pneumothorax, like which it causes distention of one side, displacement of the heart, diminished motion, tympanitic resonance and feeble or suppressed respiration with metallic tinkling." The history will often be of the greatest aid in reaching an accurate conclusion. In the cases collected by Bowditch, 26 were congenital and the rest traumatic. The principal symptoms noted were dyspnea on exertion and sometimes on lying down; pain in the bowels, especially after a full meal, in those where the opening through the diaphragm was small, and vomiting in several cases where the stomach was wholly or in part within the chest. In most of the cases the pulse was disturbed and in a few it was weak and rapid. In 27 per cent. of these cases the patients were in good health and engaged in active business.

In Abel's case the symptoms, which came on suddenly, were those of obstruction of the bowel, with vomiting, profound collapse, retraction of the abdomen and distention of the left side. He based his diagnosis upon the retraction of the abdomen, distention of the left side with tympanitic resonance and displacement of the heart to the right, with the collapse, and absence of any passages either of gas or feces from the bowels. In his case it was impossible to introduce a stomach tube through the cardiac orifice; therefore this organ could not be irrigated. In Leichtenstern's case he was able to demonstrate the presence of stomach and intestines in the pleural cavity and to note changes in them from the introduction of water and air. This patient complained of dyspnea on exercise, but worse during eating. There was slight cyanosis of lips, increased respiratory frequency and almost an absence of heart dulness. Percussion and auscultation over front of thorax yielded normal signs. Heart sounds muffled, and loudest at lower end of sternum. Abdomen flattened. Posterior part of thorax: slight loss of motion and prominence below the left shoulder blade. Percussion and auscultation normal to lower angle of left scapula. Below this a hollow, deep, tympanitic note. In this region absence of vesicular breathing; instead

*Read before the Chicago Laryngological and Climatological Association, May 2, 1901. (See Proceedings, p. 1801.)

1. Leichtenstern: *Berliner Klin. Wochenschrift*, 1874, p. 497.

2. Thoma: *Virchow's Archiv*, vol. lxxxviii, p. 515.

3. Abel: *Berliner Klin. Wochenschrift*, 1894, p. 84.

4. *Dis. of Chest, Throat and Nose*, 1900, 4th Ed., p. 88.