ment is subjected to spectroscopic examination. I have elsewhere given in some detail the reasons which show that skatol-red and the urorosein reaction do not depend on the presence of the same mother substance in the urine.⁸ Since discussing this subject I have been able to demonstrate the dependence of the urorosein reaction on the presence of indolacetic acid in the urine. When this fact is realized by the writers who maintain that skatol-red and urorosein are identical it is evident that this contention will have to be abandoned.

The view that indolacetic acid arises from the decomposition of proteids in the intestinal tract is supported by the fact that I have been able to obtain from the intestinal contents of a patient showing a marked indolaceturia a substance showing the reactions for indolacetic acid. I have not yet, however, had the time to prepare this substance from the intestinal tract in sufficient quantity to make melting-point determinations and to demonstrate the formation of skatol on subjecting the substance to a temperature in the neighborhood of 180° C., these being the criteria which serve positively to identify indolacetic acid. It is entirely in harmony with the view that indolacetic acid arises from tryptophan decomposition in the intestinal tract that I have been able to induce a strong urorosein reaction in the urines of two healthy subjects who during several days ate a large excess of beef—from 3,000 to 4,000 grams daily. Previous to the eating of the beef in excess, the urines from these subjects failed to show the presence of indolacetic acid. My explanation of this induced indolaceturia is that in both instances the presence of such large quantities of proteid in the intestine gave rise to a delay in the absorption of tryptophan formed through digestive proteolysis and that in consequence of this delay indolacetic acid was formed through the action of bacteria on tryptophan. This observation leads me to suspect that there are instances of pathologic conditions in which the excessive use of proteins is at least a factor in the development of indolaceturia. I would refer in this connection particularly to two types of cases, namely, diabetes and certain instances of chronic intestinal indigestion in children. In diabetes there is a tendency for patients to restrict themselves in carbohydrates and to compensate for that restriction by increasing the fat and protein intake. The protein intake under these conditions may be 50 or 100 per cent. greater than would be the case in health. The same thing is true of a group of cases of chronic intestinal indigestion in children in which it is impossible to take more than very small quantities of carbohydrates without excessive and detrimental fermentation. These patients, moreover, are frequently unable to tolerate fats in normal amounts. Under such conditions the physician tends to recommend a diet rich in protein.

It thus appears probable that the excessive feeding of proteins may be concerned with the development of indolaceturia. I do not maintain, however, that this is essentially the cause of the indolaceturia in the two classes of cases just mentioned. For I have seen the phenomenon in persons who are taking no excess of protein food. Moreover, in pathologic cases of indolaceturia, in which the protein food has been restricted, the indolaceturia may be still persistent. It is thus clear that, while the intake of a large amount of protein is a factor highly favorable to the development of indolaceturia, the occurrence of this condition must be regarded

as depending rather on delayed absorption of tryptophan and suitable bacterial conditions than on mere over-feeding with proteins.

Assuming that the hypothesis set forth in this paper is correct and that both indolacetic acid and the indol formed in the intestinal tract are derived from tryptophan through the action of bacteria, the fact remains still to be accounted for that sometimes the decomposition of tryptophan yields mainly indolacetic acid and at other times mainly indol. I am at present unable to offer a satisfactory explanation for this selective breakdown of tryptophan. As is well known, bacteria of the *B. coli* group are capable of clearing tryptophan to indol and, as already mentioned, it was shown by Hopkins and Cole (an observation confirmed by Dr. Dakin and myself) that these organisms are able to form indolacetic acid from tryptophan. It is doubtless true that in the intestinal tract the bacteria associated with the colon bacilli play a part in determining the direction of the main cleavage of tryptophan, and I think it possible that through the careful study of the symbiotic action of bacteria on this substance it will be possible to gain an insight into the different bacterial conditions that determine the production of indolacetic acid rather than indol.

It is not my intention here to discuss the pathologic consequences of indolaceturia. I may point out the fact, however, that, since indolacetic acid is not paired in the organism like indol and skatol and phenol, it may have an opportunity to act directly on the nervous system when absorbed in considerable quantities. A comparison of the action of indolacetic acid, both on the muscle functions and the functions of the central nervous system with the effects produced by indol and skatol, is thus a distinct desideratum. Dr. Lee, who has interested himself in the effects of indol and skatol on muscle fatigue, has kindly offered to study the effects of indolacetic acid on the living muscular substances. I have not yet had sufficient experience in the therapeutic modification of indolaceturia to express any opinion as to the best method of ridding the organism of this condition, but in any scheme of treatment the diminution of protein food would have to be considered.

Original Articles

STREPTOCOCCIC INFECTIONS OF THE PHARYNGEAL ADENOID TISSUE IN ADULTS.*

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This subject is of sufficient interest and practical importance to receive at the hands of the laryngologist much more careful consideration than it has in the past. I am not sure whether these streptococcic infections of the pharyngeal adenoid tissue in adults are thought of or even clearly recognized by the general practitioner. The basis of this paper consists of data

8. Indolacetic Acid as the Chromogen of the "Urorosein" of the Urine, Jour. Biol. Chem., 1908, iv, 253.

* Read in the Section on Laryngology and Otology of the American Medical Association, at the Fifty-ninth Annual Session, held at Chicago, June, 1908.

collected in ten cases, occurring in my own private practice.

There never was a period when, more than now, the laryngologist devoted time, ingenuity and skill to refining his instruments, extending his view of experimental and research work, and perfecting a multitude of operations. May not one think that we have been over-zealous in our activity to reconstruct the nose and to devise new ways to explore the nasal sinuses and cells, and to peer into remoter regions of the esophagus, stomach and bronchial tubes? Thereby, are we not peculiarly tempted to lose sight of the older and more familiar fields?

In 1868 W. Meyer first drew attention to the importance of the clinical aspects of the pharyngeal adenoid tissue. No subject in our line up to the present time has received more widespread attention. Next to no information can be gleaned from text-books. Though a careful research of the literature has been made, the scientific papers scattered throughout the medical journals offer but scanty material upon this topic.

Let us consider, for a moment, how sometimes the naso-pharyngeal condition may be overlooked or little importance may be attached to it. This statement may seem paradoxical, but it shows how easily we may be led astray when symptoms are misleading or simulate, perhaps, some general diseases, as pulmonary tuberculosis, malaria, typhoid fever or influenza, thus masking the original seat of the trouble. This is obviously true in cases of differential diagnoses.

It is hardly possible to overstate the importance of direct examination of the postnasal and nasal regions to settle any debated question where septic conditions are present that cannot be attributed clearly to some one definite region of the body. Another safe rule to follow is to have bacteriologic examinations made of these regions; and lastly, the thermometer might throw some light on many doubtful cases of general disturbance where the nasopharynx is the primary site of infection.

I will outline a case with obscure symptoms due to an unrecognized throat trouble.

CASE 1.—History.—Miss A., authoress, aged 30, was sent to me in November, 1904. Three months previously she had had an attack of influenza. This passed off, and the patient reported herself as slowly regaining her usual state of health. But two weeks previous to her visit there had been a rise of temperature each evening, ranging from 100.5 F. to 101.5 F., with a slight elevation in the morning. She had been examined carefully on two or three occasions for a possible pulmonary involvement, but always with negative results. It was stated that the throat felt a little sore, with a slight increase in postnasal secretion, and for a day or so the voice had become slightly nasal and husky.

In addition, a cough developed, and masses of yellowish white, opaque material, a centimeter or less in diameter, were expelled freely. The patient had noticed no bad taste or disagreeable odor, nor was it necessary to use the handkerchief more frequently than usual. At no time during her convalescent period had she felt ill enough to go to bed.

Examination.—The larynx was examined. It appeared slightly reddened. The nasal membranes were inflamed, but no undue amount of secretion was present in the meatuses, and the maxillary sinuses were clearly illuminated. The soft palate and uvula and posterior pillars and pharyngeal walls showed marked swelling and redness. The tonsils were swollen, very slightly inflamed, with no exudate or membrane covering them. Posterior rhinoscopy brought into view the adenoid tissue covered with secretion of creamy consistency. This was cleared away by a saline spray. The adenoid tissue looked "rat-eaten," and only one-third of it remained, and

this remnant finally came away with the slightest touch of the forceps.

The bacteriologic examination showed streptococci in abundance, and the masses that had been expelled were remains of adenoid tissue.

The constitutional symptoms disappeared, and the local signs cleared up promptly under appropriate treatment.

In the following case, the symptoms were not particularly misleading, but they were unusual in their severity and rapid onset:

CASE 2.—History.—A physician unduly exposed to septic infection became ill May 5, 1907. The illness was ushered in by chilly sensations and a rise of temperature to 104 F. on the afternoon of the first day. General malaise, headache and anorexia soon followed, with indefinite pains about the post-cervical region. For three or four days the temperature rose from 99.5 F., or thereabouts, in the morning to 102 F. or 102.5 F. by 6 p. m. The pulse meantime was irregular, soft and rapid, ranging from 85 to 115. The symptoms generally seemed to simulate an attack of influenza, for which it might have been taken. But at this time the soft palate was slightly painful, as were also the posterior pillars. In less than two days, the pain in the throat became very severe after using the voice or on deglutition. At night it was sufficiently severe to prevent sleep.

Examination showed the pharyngeal walls to be red and swollen. The uvula, soft palate and posterior pillars were markedly red, swollen and edematous. The laryngeal condition presented nothing beyond a general redness and the nasal condition was similar. It was only toward the end of the illness that the nose was secreting freely, which was traced to the postnasal condition. At the end of five days the pain in the throat became paroxysmal. It was slightly relieved by clearing the postnasal region, aided by very warm normal salt solution, in addition to a decided muscular effort. Later, the pain abated somewhat for half an hour, following the expulsion of pieces of soft friable tissue of a pinkish color. On the fifth, sixth and seventh days of the illness, three such pieces of tissue were expelled, varying in diameter from 2 to 3 centimeters. A fourth piece came away immediately after a post-nasal application.

Postnasal examination showed intensely swollen pharyngeal walls with a small amount of adenoid tissue remaining. At a previous examination some four years ago this patient was known to have had only a small adenoid that in no way blocked the choanæ.

Bacteriologic examination gave a pure growth of streptococci, and the microscopic examination of the expelled tissue, as reported by Dr. Elizabeth Morse, showed several pieces of opaque white, firm material, composed largely of débris, fibrin network and dense collections of pus cells. In some areas there is a delicate reticulum containing in its meshes polymorphonuclear leucocytes and a few lymphoid cells. Except for the latter, no fixed cells are seen. No blood vessels.

This case yielded promptly in three weeks to nasal and post-nasal applications and sprays and to appropriate general treatment.

In a third case where the symptoms pointed to the throat but the exact location was overlooked.

CASE 3.—History.—Mr. C., a lawyer, consulted me in January, 1906. He had been exposed to a severe case of scarlet fever in his own family. He had never had influenza and gave no history of nose or throat trouble. However, he had had a sore throat for four days previous to his visit to me. There was general malaise and marked pain in his throat, and also at the back of his neck. There was slight soreness on swallowing, and more or less blocking of the posterior nares. The patient had become nervous and irritable. There was a rise of temperature, never reaching over 102 F. in the evening, with exacerbations each day. The respirations were not much above normal; the pulse beat was accelerated, but otherwise not abnormal. Prior to coming under my care he had been treated by incisions, first in one posterior pillar and then in the other. Finally, the tonsil was incised at its apex in the

hope of finding deep-seated pus. This procedure proved fruitless and only increased his discomfort.

Examination.—I saw him a day later, when there was most extensive swelling of the uvula, soft palate and posterior pillars. The tonsils were only slightly reddened. Anterior rhinoscopy brought into view reddened mucous membrane. There was no secretion in the meatal recesses and the antra showed up well under illumination. An examination of the postnasal region showed a yellowish white mass in the region of the adenoid. A very warm saline spray was used, when friable pieces of material one-fifth of a centimeter in diameter came away freely, leaving the base of the adenoid very uneven in outline.

This case was treated similarly to the others, and within two weeks the throat symptoms had subsided. It may be well to note that I had seen this patient some three years ago, when he was referred to me for the removal of impacted cerumen. At this time, it was recorded that the adenoid tissue was small in size.

In presenting these cases I have reported a sufficient number to illustrate the points I desired to note; and these three cases were the most pronounced of all. I am convinced that such cases are likely to be comparatively rare. In addition to the above there are seven others on my records, but to detail them would be only a repetition of the foregoing, with symptoms very much less marked. I may mention that the ages ranged from 30 to 50 years. In no case were the ears involved, nor was there an enlargement of the cervical glands. All the patients recovered.

In this brief paper no attempt has been made to go into exhaustive details of the cases, or to approach the subject otherwise than as met with in adult life. To study this subject in infancy or in early years might well furnish material for special papers. However, incomplete as it is, it may further in other workers more searching and fruitful investigation.

It needs no homily to convince us of the value and importance of this subject in the rôle played by the streptococcic infections of the pharyngeal tissue, not only in their local, but also in their remoter effects, whether the symptoms are definite or obscure.

My best thanks are due my colleague, Dr. Katharine H. Law, who kindly looked over the literature of the last fifteen years.

DISCUSSION.

DR. B. R. SHURLY, Detroit, said that the serious sequelæ and complications of the infectious diseases, such as influenza, typhoid fever, diphtheria, scarlet fever, measles, tonsillitis, arthritis and others are in direct ratio to the absorption of toxin from the nasopharyngeal space. This pathologic and therapeutic principle is not sufficiently recognized by the average general practitioner. Acute infections of the lymphoid ring, especially those of the streptococcic variety attended by high temperature, myalgia and glandular swelling, naturally fall within the realm of bedside medicine. In private practice it is rare that in acute cases of this type the patients are subjected to rhinoscopic examination; unless throat trouble is evident on a simple inspection, other and more remote regions of the body are investigated. If complications arise, the internist or pediatricist is called, as the laryngologist of the last decade is usually a surgeon and not a clinician. Dr. Bryant's plea for a return to the older and more finished field is a plea for more laryngologists who have been trained in the hard and fascinating school of general practice. To carry out this examination satisfactorily at the bedside, as a routine measure requires more inconvenience than the busy internist will suffer. From the view point of the rhinologist, however, no examination could be complete without posterior rhinoscopy. The three cases reported are types of acute infections that occur in enormous numbers through the winter and early spring of our northern climate. They are classi-

fied popularly as influenza. Depending on innumerable factors such as the variety of bacteria, virulence, age, resistance of the patient and area of lymphoid tissue infected, these epidemics are attended by a train of similar symptoms. The clinical picture changes in relation to the bacteriologic findings. Streptococcus and pneumococcus in pure culture rank second only to influenza as serious complication. Dr. Bryant reports no involvement of the middle ear in her series of cases, although this particular variety of infection is extremely liable to extension. The cases described gave evidence of acute inflammation in the throat as well as the adenoid involvement. There was streptococcal infection while the individuals were in a state of so-called lowered resistance from various causes. Case 1 illustrates a type that is frequently classified as recurrent influenza by the general observer and which may be scientifically classified as chronic influenza in many cases. This condition is a distinct entity as a pulmonary disease, and is differentiated with great difficulty by physical signs and symptoms until a special stain reveals the influenza bacillus. Acute inflammations or chronic inflammations of the lymphoid ring due to this bacillus lead to marked susceptibility of the mucous membrane to mixed infection with pyogenic micro-organisms. A chronic influenza of an adenoid may exist for weeks after an attack that is looked on as a simple nasopharyngitis. Cases 2 and 3 illustrate frequent types of acute infection that follow work in the postmortem room or among septic wards of hospitals. Those who have served at length in the contagious departments, especially among scarlet fever patients, can recall many cases among physicians and nurses of streptococcal infection. They are usually of attenuated forms and respond readily to treatment. Dr. Shurly agreed with Dr. Bryant that in acute infections of the type under discussion, the old time spray of Seiler's or Dobell's solution or warm boric acid solution is exceedingly grateful and beneficial to the patient. Gentle irrigations of salt solution or compound antiseptic powder U. S. P. with a post-nasal syringe are useful. While many cases respond promptly to irrigation, sprays and internal medications, a few persistent cases of streptococcal infection of the pharyngeal adenoid and adjacent structures may require the aid of bacterial vaccines. In Dr. Shurly's experience it has proved of value in several threatening cases of erysipeloid variety. Stock vaccine may be used when time or inadequate laboratory facilities will not permit the more successful opsonins to be prepared for the individual, and the proper treatment of acute infection in this region will limit the complications and therefore the surgery of the sinuses. Prevention of acute adenoiditis by the removal of the diseased gland or other prophylactic measures demand more universal attention. Dr. Shurly asked Dr. Bryant if these cases had been investigated by culture methods, especially for the Pfeiffer bacillus.

DR. EMIL MAYER, New York, said that adenoid vegetations in adults are far too frequently neglected. He emphasized not only that this infection occurs in the adult, but also that the adenoid vegetations should be more frequently recognized than they are. We have not only this class of infections in the adenoid tissue, but also a direct inflammatory affection which physicians have been treating for years as ordinary coryza, ordinary pharyngitis, or an ordinary tonsillitis, perhaps only treating the pharynx and neglecting far too frequently the presence of adenoid vegetations. He has a number of adult patients with adenoid tissue quite as large as that found in young children.

DR. J. E. LOGAN, Kansas City, declared that there is no doubt in his mind that physicians have failed signally to consider seriously the adenoid in the adult, that may of itself appear absolutely innocuous. It may be so small in size that it may readily pass unnoticed. For that reason operative procedures that will reach those conditions that are so manifest in the vault of the pharynx, and so manifest in all the infectious diseases of the nasopharynx and of the tonsillar cavity are neglected. His experience shows that most cases of otosclerosis that have been so particularly obnoxious to the practitioner, to the laryngologist or to the otologist have originated

in this neglected condition. He believes that in many of these cases that have resisted all medical treatment previously, and in which there is the slightest evidence of any vegetations on casual examination, those little creases and sulci and pharyngeal recesses that are so prevalent in all of these adults can be found in the vault. This little remaining fibrous condition is of the greatest importance in the relief of all conditions in the middle ear, chronic catarrhal conditions, and the many infectious diseases found in the nasopharynx. The crises, as suggested by Dr. Mayer, are likely to recur, and this tissue is a ground for the culture of all infectious material. Dr. Logan has been able to make numerous investigations along the line of the vault of the pharynx in these apparently indifferent cases of all classes of bacteriologic and pathologic manifestations, and has found the Klebs-Loeffler bacillus, streptococci and staphylococci.

DR. ALICE G. BRYANT, Boston, in reply to Dr. Shurly said that these ten cases were not investigated for Pfeiffer's bacillus.

LARYNGEAL MANIFESTATIONS IN LOCOMOTOR ATAXIA AND MULTIPLE SCLEROSIS.*

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The astonishing findings of Dr. W. B. G. Harland in cases of locomotor ataxia caused me, after reading last year's preliminary report of the Section on Laryngology and Otology, to examine all such cases at my disposal at the Montefiore Home, with the view of presenting the results in the subsequent discussion of Dr. Harland's paper. Unfortunately I was prevented from attending that meeting, but the investigations were not abandoned; on the contrary, they were repeated. Afterward, all the cases of multiple sclerosis were also examined, with the view of clearing up some pending questions, especially in regard to paralysis of the abductors, the modifications of the voice, the ataxic movements of the cords, etc. Finally, a visit was made to the City Hospital on Blackwell's Island, and fourteen cases of tabes were examined. But I do not consider these last findings of absolute statistical or scientific value, for the reason that the patients were examined only once, which can not be considered a sufficient basis on which to pass final judgment in locomotor ataxia.

The statements of Dr. Harland that "the larynx is very seldom seriously affected in locomotor ataxia," and that he was unable to find a tabetic case with laryngeal crises, were so much at variance with my own experience and all that I had read on the subject, that I naturally devoted especial attention to these points.

In order to make it easier to understand these cases, their histories should have been given in full, but they had to be abridged or this paper would have been too lengthy. The histories of the cases examined only once at the City Hospital were purposely omitted. The same has been done, to a large extent, with the cases of multiple sclerosis, as in this connection they are of secondary importance, although highly interesting in themselves.

CASE 1.—*History*.—Samuel W., aged 50, admitted June 6, 1907. Chancre at age of 20; used tobacco in excess. Present illness began twenty-five years ago, with shooting pains in both legs. One year later right arm suddenly became paral-

alyzed. For past fifteen years has had trouble with his bladder and rectum; three and a half years ago fell and fractured left patella. June 18, 1907, fractured left tibia from a very trifling cause.

Examination.—Jan. 30, 1908. When patient speaks has to stop often on account of tickling in his throat. Has a marked postnasal catarrh with a good deal of secretion. Right vocal cord fixed in the median line. Left vocal cord moves about 2 mm. on inspiration. Secretion on the right vocal cord. False vocal cord thickened. No dyspnea. February 13: Right vocal cord fixed in median line, border straight; left vocal cord moves very little; voice gives out easily.

Diagnosis.—Paralysis of both postici, with secondary contraction of adductors of right side.

CASE 2.—*History*.—Harry S., aged 43, admitted March 23, 1903. Present illness began five years ago, with dizziness and shooting pains. Three years ago a general eruption appeared; was given a thorough course of inunctions, lasting four months. Girdle pains then developed; patient had to be catheterized and began to have difficulty in walking. Vision failed and paresthesias in the feet appeared. Developed a perinephritic abscess, which had to be opened at Montefiore Home. Has frequent gastric crises. No laryngeal crisis.

Examination.—April 15, 1907: Larynx normal. Jan. 27, 1908: Can speak in a loud voice; right vocal cord almost immovable in median line; left vocal cord moves to "cadaveric" position on deep inspiration.

Diagnosis.—Complete paralysis of posticus on right side; partial paralysis of left. The progressive character of tabes was noticed in this case. In April, 1907, the larynx appeared normal, while a year later there were marked lesions of the motor apparatus.

CASE 3.—*History*.—Mrs. R. G., aged 49, admitted April 1, 1903. Married and had four children, of which the first born was marasmic; then a number of abortions; only one child is living. Had cataract at age of 26, since which time she has had trouble with her vision, until finally she became blind. About ten years ago had shooting pains in the lower extremities; two years later paresthesias in the soles of the feet. About this time locomotion became impaired. April 5, 1899: Was attacked by dyspnea, cough and severe hic-cough which lasted about ten minutes. She then became cyanotic and artificial respiration was resorted to. Oct. 11, 1904: Again had laryngeal crisis; cyanotic for about ten minutes.

April 29, 1907: For three days patient has had severe shooting pains in her left loin. Yesterday uncontrollable vomiting and retching. Just at noon to-day patient suddenly became cyanotic after coughing for a time, breathing very stertorously and in gasps; she soon became unconscious, frothing at the mouth, and then lived and almost cadaveric. Except for an occasional gasp, breathing ceased. Venesection was performed and eight ounces of blood abstracted; meanwhile artificial respiration was kept up. In about ten minutes she regained consciousness; following this she had hic-coughs for four hours. Larynx normal.

Diagnosis.—We have here an exceptionally severe example of laryngeal crisis, which reminded the writer of a case reported by Cherchevsky. The attack observed by him was very similar to the one described above and lasted forty-eight hours (!). The patient recovered, but died suddenly of a similar seizure several years later.

CASE 4.—*History*.—Mrs. R., aged 61, admitted Aug. 30, 1904. Husband died of dementia paralytica. Twenty years ago had attacks of severe vomiting of obscure origin, at times accompanied with diarrhea and fainting; sixteen years ago shooting pains and paresthesias. Soon after, patient experienced difficulty in walking. Had diplopia ten years ago. Had frequent gastric and, at times, rectal crises; no laryngeal crises; deglutition normal.

June 21, 1907: Partial paralysis of right vocal cord.

Examination.—Jan. 24, 1908: On phonation, vocal cords do not close in median line, a straight narrow slit of about 2 mm. being left between them. On inspiration vocal cords do not open to their full extent.

* Read in the Section on Laryngology and Otology of the American Medical Association, at the Fifty-ninth Annual Session, held at Chicago, June, 1908.