LARYNGEAL VERTIGO.*

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On December 5, 1896, a gentleman, Mr. W. L. Wright, consulted me concerning a cough from which he had suffered many years, but which lately had been attended with alarming symptoms.

He was aged forty-seven, weighed 173 pounds, and was five feet four inches in height. His complexion was florid, his hair quite gray, his manner placid and temperament phlegmatic. His pulse was ninety, a trifle irregular, small and compressible. His urine acid, specific gravity 1.029, loaded with phosphates. He led a quiet, regular and sedentary life, being curator of a large estate; did not use tobacco or alcohol, took extremely little exercise, but ate largely of nitrogenous foods; meat three times daily, especially beef, beef-steak and mutton, eggs in abundance, large quantities of bread and potatoes and cooked fruits. His flesh was soft and flabby; his chest narrow, but abdomen large and pendulous. All his visible mucous membranes were equally hyperæmic, that of his larynx not more so than the gums or conjunctiva. He was plainly in a condition of what I, for convenience sake, style hypernutrition. For years his bowels moved five or six times daily, the amount, however, in toto not exceeding one full evacuation, as is his habit to-day. He also passed urine very frequently, but not in large amount. Commencing at the age of seven, after an attack of scarlet fever, he had been the subject of winter cough, which almost completely disappeared with warm weather, or, as questioning elicited, when his skin began to act more freely. This cough was paroxysmal, sometimes but two or three attacks in a day, sometimes twenty, the paroxysms ending with the ejection of a small amount of viscid white sputum, which had never become yellow, therefore not of inflammatory origin—not a bronchitis, but the result of bronchial hyperæmia of varying intensity. For the last twenty-five years he has been the subject of hay fever, and for the last nine years, during the latter weeks of the hay fever season, of asthma.

From this history it was apparent that in addition to his state of hypernutrition there was defective elimination, and that the principal irritant in the blood was uric acid.

In October and November of 1895 he noticed that on several oc-

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casions his fits of coughing were accompanied or followed by vertigo, so that frequently he, for a few moments, clung to the nearest support. In December of that year, whilst seated in the train on his way to his home at Webster Groves, he suffered one of his usual cough paroxysms, and found himself cold and his clothing quite wet. He asked his companion in the seat for an explanation, and was told that he had fallen over on him, ceased to breathe, became very red in the face, had excited alarm about him, and had been drenched with ice water to restore consciousness. In this winter, that of 1895-96, he had three more such attacks. For the three months preceding his visit to me (December 6, 1895) he would return home from his day's work quite exhausted and somnolent, so that if, for example, supper was not quite ready, he would fall asleep sitting upright in a chair. He had also had, with hardly exception, every evening, between six and nine, sometimes at the supper table, usually afterward, an attack of cough, followed by complete unconsciousness. For a description of an attack I am indebted to his very intelligent wife. No action in particular attracts her attention, but experience has taught her and her daughter to watch him carefully. She notes first a peculiar, indescribable facies, his eyelids dilate, the eyeballs roll upward, become dull and listless. This facies lasts, sometimes, a full minute or two, long enough, frequently, to enable them to drag him, in his chair, to the window, which they open and, in addition, vigorously fan him. She thinks him semi-conscious, and by this treatment has been enabled to ward off the next phenomena, which consists of, almost invariably, three short, dry coughs, each lower in pitch than the preceding one, followed invariably by complete unconsciousness. He ceases to breathe, his face becomes bluish-red, his whole body becomes somewhat rigid, and thus he remains from five to twenty seconds, when a long inspiration takes place and the attack is over, except that on a number of occasions there has followed, for two or three minutes, mental confusion. These attacks have very seldom followed violent coughing, but she has never known him to have an attack without the preceding three gentle dry coughs. There has never been an involuntary cry, escape of urine or biting of the tongue, or convulsive movement. Several cases have been reported in which there was first a laryngeal or sub-laryngeal tickling, but my patient states that he has never experienced this or any other morbid sensation in the throat at any time. His family history is perfect, and beyond the history I have given his health has been excellent. Physical examination yielded no evidence of disease in any part of the body. At most, there was a gentle hyperæmia of

the upper respiratory organs. In accordance with my theory of the pathology of his case, namely, that there occurred uric acid explosions, producing either capillary spasm or paresis in the respiratory centers, thus in turn producing not only spasm of the glottis, but, as I wish to insist on, spasm of all of the muscles of respiration. He was placed upon anti-lithaemic treatment, the details of which it is of no import to mention here. It consisted greatly of rigid diet, cold friction to the skin, calisthenics, and the substitution for a certain amount of intellectual work, manual labor in the open air. By February 22 he had lost nineteen pounds, and yet had gained quite considerably in strength of mind and body. He had but two movements from the bowels daily, instead of six; his urine was straw-colored and transparent; his asthma had disappeared; he coughed fifty per cent less than he had in twenty years, and it had lost its dry, paroxysmal nature, the sputum being ejected without effort. He had, from December 6th to the 15th, but two of his laryngeal attacks, and since that time not one. He was discharged from treatment, but instructed to so diet and exercise as to maintain the muscular equilibrium he had secured. I did not see him again until October, 1897. He had been perfectly free from attack, and related an interesting history bearing upon the diagnosis of the uric acid diathesis. For many years his hay fever had invariably commenced on August 16, but he had always, for a few days before, had certain prodromata. Last summer he did not leave for his annual vacation until August 21, and up to that date had not even had the usual hay fever prodromata. Unfortunately, for the sake of this history, he went to his usual resort in Canada, one which is exempt from hay fever, so that whether he would have escaped his hay fever or not, cannot, of course, be stated.

I did not see the patient again until October 8, 1897, when he applied for relief from a mild attack of subacute bronchitis, which yielded very quickly to an apomorphia expectorant mixture.

I have seen him again lately. He states that he has not had a single attack of cough, followed by unconsciousness, but that he has had several mild attacks of vertigo following cough. He has been overworked, has neglected his calisthenics, but has maintained the reduction in weight. He still has remnants of his old winter cough, but has suffered no attack of the peculiar spells of short dry coughs which have preceded his attacks of unconsciousness.

Since 1876, when Charcot first drew attention to the clinical picture, of which my case is a reproduction, about fifty cases have been reported. The salient features are, however, so alike in all reports,

the group of symptoms so peculiar and characteristic, that there is formed a distinct morbid entity, and yet not entitled to any rank in nasology. Baptized by Charcot laryngeal vertigo, the title can only be accepted as one of convenience, since vertigo is but a symptom of many diseases, and the larynx in the majority of cases has been found absolutely normal. In a few cases there existed laryngeal hyperæmia, and in but one case, that of Sommerbradt, all symptoms vanished subsequent to the removal of a laryngeal polyp, but in this same patient, fifteen years before, epileptic attacks had ceased after the removal of a cicatrix from the dorsum of the hand.

The essential difference between this disease and the laryngeal crisis of locomotor ataxia is in the cardinal laryngeal symptom. In tabes the cyanosis and unconsciousness only prevail after the glottic spasm has existed quite a time, just about the same length of time as would arise from ordinary strangulation, whereas in laryngeal vertigo the unconsciousness and cyanosis appear immediately after the laryngeal symptom, be it cough or laryngeal tickling, or both, so quickly, indeed, that mere glottic spasm cannot alone explain the vertigo or unconsciousness.

The attack so often resembles *le petit mal* of epilepsy that several authors, notably Gray and Schrötter, describe it under that title. Half the number of patients have laryngeal or sub-laryngeal tickling—a sensory aura—whilst all, with hardly an exception, have two or three very gentle cough explosions immediately preceding the loss of consciousness—a motor aura—yet there are some notable differences. Out of the fifty-three cases reported, but three have been in females; there has been marked absence of heredity, and in nearly all I know of no case reported in children.

All cures have quickly followed various therapeutic measures. In a very few the tongue has been bitten, and there have been slight convulsive movements, but never true clonic spasms; never involuntary passage of urine; never the epileptic cry; never part epileptic phenomena, except in a few cases a slight and transient mental confusion, so that there exist but the two symptoms common to both, an aura, quickly followed by insensibility, a clinical picture which has appeared after douching an ear, or the nose, or adding another epileptic sign; convulsions have been seen in laryngismus or whooping cough.

Out of seven cases collected by Schadowaldt, of Berlin, six were pronounced alcoholics, and in most cases reported the patients have been plethoric. Schadowaldt also reports the only death, and is one of the very few who has been an eye-witness of the attacks. He

relates the difficulty of analyzing the symptoms, because the whole attack is so quickly over. He has seen this patient, as I have mine, in tremendous paroxysms of cough, and yet not followed by insensibility. At most there was but slight vertigo. The disease in others, however, has followed violent explosions of cough. In the lethal case of Schadowaldt there was but slight spasm, the usual gentle cough, insensibility and death. The disease had disappeared in this case for twenty months. The author calls attention to the fact that perhaps there are many more deaths than are reported. Suppose, for example, this patient, after recurrence from what had seemed cure, had, after relapse, applied to some other physician who had not recognized the character of the case, or had he been found dead, the ultimate outcome might never have been known.

What, then, is the mechanism of events in laryngeal vertigo, or as most authors now call it, *ictus laryngis*, since it is not with that stage or symptom of the trouble—vertigo—with which we are dealing, but where there is a stroke, implying insensibility?

There are a few expiratory outbursts, namely, the gentle coughs, and in this attitude, that of a closed glottis, respiration instantly ceases. Not only is there addular laryngeal spasm but there also occurs that which no author seems to have mentioned, namely, spasm of the great abdominal muscles and the diaphragm in the expiratory attitude. Whether there is simply glottic spasm, or with it associated spasm of the other respiratory muscles I have mentioned, makes a very great difference in the intrathoracic pressure. If any one of my audience will take a deep breath and then simply hold it, he will find that the face becomes turgid very slowly, that the pulse will become slower, but hardly change in volume, whereas let him take a deep breath and then throw his diaphragm and great abdominal muscles into expiratory spasm, the glottis will close, his face will at once become turgid, the pulse slower and feebler and finally disappear, and syncope may occur. These experiments were first described by Weber in 1851. On one occasion, after self-experimentation, he actually became unconscious, there were muscular twitchings in his face, and some mental confusion.

Dr. F. T. Knight, of Boston, in an article on laryngeal vertigo, published in 1886, recalling these experiments, following McBride, explains the symptoms by saying that "there is a direct disturbance of the cerebral circulation by compression of the large blood vessels and even of the heart itself." He recalls that wind-instrument performers often suffer vertigo—that rapid respiration may cause sufficient anæsthesia to admit of the painless extraction of a tooth.

Charcot makes it a condition analogous to Meniere disease and stomachic vertigo. He reminds us of the experiments of Paul Bert, where, through irritation of the pneumogastric, respiration and other motor phenomena may completely cease and death follow. He concludes that the upley cycle is contripetal, and the starting point some irritation of the superior laryngeal nerve at its distribution in the larynx. Just as in hay fever the sensory irritation is in the nose. In the one case the resultant motor result is glottic spasm and in the other sneezing. But I wish to add that just as in hay fever the efferent impulse may be conveyed to the bronchi and asthma result; so in laryngeal vertigo—the efferent impulse may produce not only glottic spasm, but also spasm of the great abdominal muscles and diaphragm.

Against Charcot's explanation may be mentioned the fact that in these very patients it is exceptional to find the attacks follow violent attacks of coughing, and also the clinical fact observed by the wife of my patient that certain phenomena preceded the laryngeal tickling, as the gentle explosions of cough, which precede the unconsciousness. She observed a certain facies, the dilatation of the lids, the upturning and glazed expression of the eyes, to precede the cough. As I have already stated, no medical man except Schadowaldt has had the opportunity of witnessing and analyzing the attack. It may be found that if those who see the patient in the attacks be instructed to look for the facial expression I have mentioned, that it may always be found to precede the laryngeal tickling.

My own view is that the first phenomena is central, most probably a vaso-motor spasm about the respiratory center, and that it is the central irritation which causes first the laryngeal tickling or short, dry coughs, and then the glottic spasm, as well as that of the great abdominal muscles. The insensibility follows instantly the glottic spasm and cannot therefore be due to the cerebral turgescence caused by the intrathoracic pressure. In a word, therefore, I believe that all the symptoms are caused by a functional disease of the respiratory center, and in my patient, at least, from uric acid storms about the vagus centers of a man suffering nervous exhaustion.