urine was normal. (The urine had been examined on several occasions since her attack of nephritis 20 years ago and had always been normal.) During that day she had the same diet and felt better, sleeping pretty well. On the next day she felt still better in the morning and had some bacon for breakfast and some chicken at her midday meal. During the afternoon, however, the headache returned and gradually increased in severity, and she became drowsy but did not have continuous sleep. On the 15th the headache was "crushing" and she was quite drowsy and apathetic. On the 16th and 17th she was constantly sick and the bowels did not act; she could only take fluids. On the 18th she became slightly delirious, and was wandering all night. Her bowels acted well after a small dose of calomel; the urine was still normal. On the 20th she was practically unconscious, but could be roused occasionally; Dr. R. Hutchison kindly saw her with me, and he gave a very bad prognosis, expecting she would not survive many days. From then until Dec. 11th she never recovered consciousness, on some days being so deeply comatose that she would not respond to any stimulus, food placed in her mouth remaining unswallowed. For three days (Dec. 2nd to 5th) there was well-marked Cheyne-Stokes respiration. On those days she vomited several times, the vomit being very acid; she was not quite so deeply unconscious afterwards, and with much care about three pints of nourishment were able to be administered daily. On Dec. 10th she began to be less deeply unconscious, gradually improved, and made an uninterrupted recovery, being able to sit out of bed by the 20th and to go downstairs on Christmas Day, and now, a year afterwards, she feels as well as ever she did, except perhaps a little less vigorous.

The diagnosis of this case was difficult. It was evidently a toxæmia and appeared to rest between uræmia, an intestinal infection, and acidosis, but at first there was nothing to enable a differential diagnosis to be made. The urine, which was examined nearly every day, gave no definite reaction until Nov. 22nd—13 days from the onset of the condition. Then diacetic acid was found to be present and the condition was shown to be one of acid intoxication, and a line of treatment was framed to meet the condition. In order to keep up the store of glycogen the diet was entirely limited to carbohydrates, and with a view of neutralising as far as possible the acid condition bicarbonate of soda was introduced into the system as freely as possible. It was given in every feed and by slow introduction into the rectum, according to Murphy's plan, of continuous rectal infusion as long as the bowel would tolerate it, and afterwards by occasional enemata.

Bearing in mind the striking results obtained by Young and Williams, which have proved that acetonæmia varies with the amount of oxygen intake during anæsthetisation, oxygen was administered freely day and night, and one of the first recollections of the patient is the extremely refreshing sensation produced by its inhalation. Strychnine was commenced when the heart began to fail, and was kept up until recovery was assured; 1/60th grain was given every four hours at first, and subsequently twice a day. The administration of food was extremely difficult, and the patient's recovery must be largely attributed to the great care and patience exercised by her daughter and the nurse, 20 minutes frequently being required to adminster a few ounces of food.

The consideration of the etiology of this case is interesting. The exciting cause appears to have been the extreme cold experienced on Nov. 9th. This probably so interfered with the digestive processes that an intestinal toxæmia was induced; and subsequently, by their work in dealing with the resulting toxins, the liver cells were so damaged that they were incapable of performing their normal function of transforming the carbohydrates into glycogen. Moreover, as this particular patient has always been a very small eater, and is always pale and has a very small amount of adipose tissue, it is possible that her store of glycogen is always very small; and as extreme cold is one of the causes which produce loss of glycogen, it is probable that this store of glycogen became exhausted during the first few days of her illness. This view is supported by the fact that, after a temporary recovery, she became rapidly worse when acid-producing foods were introduced on Nov. 14th. The original toxæmia appeared to be passing off, but in consequence of the impaired action of the liver cells and the exhaustion of her store of glycogen, the fatty acids were not fully oxidised, and the organic acids, combining with the alkaline bases of the blood, reduced its alkalinity until the "acidosis" was produced.

The diacetic reaction disappeared from the urine on Dec. 9th and the urine has remained normal ever since. She is now taking her ordinary diet and leading her usual life.

Great Berkhamsted.

THE USE OF THE FARADIC CURRENT IN THE TREATMENT OF PERSISTENT APHONIA FOLLOWING LARYNGITIS; TWO CASES.

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IN setting forth the following cases, together with such brief comments as seem to me desirable, I have no wish to pose as a laryngologist. It is rather as one interested in the application of electricity in its simpler forms to the exigencies of everyday practice that I venture to hope what I have to say may prove of some value.

CASE 1. *Aphonia of 12 years' duration.*—The patient, a clerk, aged 38 years, in his early manhood had been what is popularly known as a "local preacher," and for years indulged in pulpit oratory of a vehement nature. On two or three occasions during this period he lost his voice for a few days, but it always returned with rest. At length, however, being upon one occasion, in the depths of winter, called to preach at a remote country chapel, he caught a severe cold and was confined to bed for a fortnight. Meanwhile his voice left him entirely, and to his alarm did not return with the subsidence of the bronchial symptoms. During the ensuing decade the patient submitted to a variety of treatments at the hands of various medical men—sprays, gargles, lozenges, internal paintings, external blisterings—in fact, ran the whole gamut of orthodox remedies many times over. All this was of no avail. Finally, at unqualified hands he underwent a four months' course of what, so far as I could gather from his description, must have been the galvanic or continuous current. The result was likewise *nil.* A laryngoscopic examination showed the vocal cords to be slightly thickened and pinkish in colour. On attempting phonation they failed to approximate fully, remaining separated by an elliptical opening—a state of affairs due to paresis of the internal tensor muscles. Judged by the long history, the case did not look very promising from the point of view of treatment. All I could tell the patient was that a course of electricity might be of benefit. He, however, demurred, saying that he had already undergone (and paid for) such a course. It was not until I convinced him by personal application that the form of current I purposed using differed from the one previously experienced that he consented to a fortnight's trial. Two weeks' mild external faradisation produced the following result. Aphonia was no longer absolute; true voice sounds could be produced at times, especially in the morning when the patient was fresh. With the laryngoscope an attempt to phonate was seen at first to be successful, the cords approximating fairly well; but the adductor muscles were not yet equal to sustained effort, and soon relaxed. A month's further treatment on these lines was carried out, with the addition of piano-aided exercises in producing and sustaining low notes. By the end of this time speech had become entirely vocal, except at such times as the patient was very fatigued, when a relapse occurred to the old whispered speech. It would be idle to say that his voice was perfect, or even pleasing, but at least he was now able to make himself heard across an ordinary room—a feat of which he had been incapable for nearly 12 years. Whether or not anything more could have been accomplished I am unable to say, as the patient's firm chose this particular time to remove him to another town. He called on me six months later, and his voice was then practically the same as when I last heard him.

CASE 2.—The patient, a school teacher, aged 22 years, a cousin of the man referred to above, came to me complaining of similar throat trouble; the aphonia, however, was not

absolute, speech in a hoarse, croaking voice being occasionally possible. There was no history of excessive strain, but one of repeated neglected "colds," with accompanying laryngitis. A gradual deterioration of the voice resulted from this neglect, and it finally became so bad that the girl was obliged to resign her position as a teacher. This occurred two years before I first saw her. A view of the larynx showed the vocal cords somewhat swollen and congested. Efforts to speak resulted in a fair degree of approximation, except that a small triangular space remained at the back of the glottis. The aphonia was therefore apparently as much due to catarrhal changes in the tension and vibration of the cords as to neuro-muscular defect. In addition to the laryngeal inflammation, there existed permanently swollen tonsils, congestion of the pharynx, and adenoids. The patient was quite unable to breathe through the nose. The question of treatment presented difficulties. The girl expected to be cured, as her relative had been, by the simple application of electricity. A consideration of the facts of the case made me not very hopeful as to the benefits to be derived from faradisation alone. I recommended preliminary removal of the tonsils, &c., but finally yielded to her importunities, and consented to try the effect of a fortnight's treatment. Some improvement certainly resulted, but it was not very striking. At the end of the agreed period I again urged surgical interference. This time the patient consented; a very thorough operation was performed, and, perhaps for the first time in her life, she was able to breathe fully through her nose. After about a week's time—that is to say, as soon as the irritation due to the surgical trauma had subsided—the voice began to improve, and continued to do so for some weeks. Two months after the operation the patient could speak vocally, but in rough, unpleasant tones. Tested by the piano for musical notes, she was unable to produce anything higher than an indifferent "middle C." I waited another six weeks, to see if any further spontaneous improvement would occur. As it did not, I decided to recommence faradic stimulation, with a view to improving the pitch and timbre of the voice. The results were most gratifying. In a short time the patient was able to manage "F," and later "A," but beyond this point no progress seemed possible. The patient's speaking voice is now of good volume and no more than slightly rasping. She has been able to obtain another post as teacher.

Remarks.—A study of the above cases, and of others of a similar nature which have come under my notice, has led me to form certain conclusions regarding the pathology and treatment of persistent post-catarrhal aphonia. Text-book accounts of chronic laryngitis and its accompanying disturbances of voice appear to me to err in regarding the disease as if it were on all fours with, say, a tracheitis. In other words, attention is concentrated on the mucous membrane to the neglect of other equally important structures. The larynx is primarily a mechanical device for the production of vocal sounds, and depends for its proper functioning even more upon the integrity of its muscles and nerves than upon the healthiness of their covering. In chronic catarrh of this organ the muscles become secondarily affected, and the delicate terminals of the motor nerves in all probability undergo an inflammatory degeneration. An acutely inflamed mucosa doubtless demands local sedative applications; and even when the trouble has become chronic, astringent sprays, paintings, &c., can often play an important part. But the tendency in many chronic cases is for the mucous engorgement to disappear to a great extent, whereas the damage to the neuro-muscular apparatus remains. Under such circumstances it is unreasonable to expect a cure by the ordinary means. On the other hand, improvement may be looked for from such measures of "natural therapy" as produce benefit in similar pathological conditions elsewhere—e.g., in that form of facial palsy which is the result of exposure to cold.

In treating paresis of laryngeal muscles by faradisation we are at a disadvantage as compared with the same conditions occurring in an arm or leg. We cannot single out the affected muscle or muscles for individual stimulation. This difficulty is not, however, insurmountable. The secret of success lies in the regular and persistent use of *mild currents*, which are not calculated to produce violent contractions of opposing healthy muscles. If carefully applied for a prolonged period this form of electricity exercises a selective action on the affected structures and eventually restores their tone.

Remarkable as are the results of faradism in suitable cases, we must not fall into the error of regarding it as a panacea for each and every form of catarrhal aphonia. Where the laryngoscope shows marked swelling and congestion of the cords the mucous membrane must be attacked vigorously on orthodox lines, and here again we are likely to meet with little success if the naso-pharynx is in an unhealthy condition. Nevertheless, even where the mucosa is the part most at fault, the judicious use of the interrupted current forms a valuable adjunct to routine treatment.

In conclusion, it is perhaps desirable to urge the necessity, in any given case, of making sure that the aphonia has really resulted from such simple causes as catarrh, overstrain, or a combination of both. Thoracic aneurysm, lesions of the central nervous system, malignant growths, the ulceration of syphilis or of tubercle—these must always be borne in mind as sinister possibilities, and to waste time in faradising a patient who subsequently dies from cancer or is cured by iodide of potassium is to run the risk of being dubbed—not undeservedly—a quack.

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NOTES ON A CASE OF PHLEGMASIA ALBA DOLENS, FOLLOWED BY PULMONARY EMBOLISM AND INFARCTION OF THE LUNG; RECOVERY.

BY J. C. CURTIS, L.R.C.P. & S. EDIN.

THE patient, a married woman aged 28 years, a primipara, gave birth to an eight months' female child on May 16th. She resided in the country and was attended by a village nurse. She went on satisfactorily until May 21st when she developed phlegmasia alba dolens in the left leg. The whole limb was considerably swollen, the swelling extending as high as Poupart's ligament. No thrombosed vein could be detected in the thigh or leg. It was therefore apparent that the circulatory block was in the pelvis. The swollen limb measured 1 inch more than the sound one, and was accompanied by the usual pain. The temperature was 101° F., pulse 90, and respirations 30. The limb was treated by lightly applying cotton-wool, and the foot of the bed was slightly elevated on wooden blocks. The lochia were somewhat offensive, and so hot permanganate douches were given night and morning. The bowels were regulated by enemata. The patient progressed favourably. On the 26th the leg was much less painful and her temperature normal, but there was no appreciable diminution in the swelling. On the night of the 28th she was seized with violent pains in the left chest and shortness of breath. As her husband expressed it, "she had awful pains in her chest, and I expected her to die every minute."

When seen on the morning of the 29th the patient was in a very distressed condition. The respirations were 72; pulse 108, regular, but very feeble; and temperature 100°. The extremities were cold. She complained of intense pain over the epigastrium, precordia, and lower axilla. She was too ill for any detailed examination, but an impaired note was found over the lower part of the axilla, and the breath sounds over this area were badly transmitted; no friction sound was heard. A soft systolic murmur could be heard over the pulmonary area and the second sound was accentuated. She suffered from retching and some vomiting, and the giving of the smallest amounts of liquid nourishment caused great pain over the precordia and lower axilla. As her stomach was dilated this was attributed to pressure on the diaphragm interfering with cardiac and respiratory movements. Hot water bottles were applied to the feet and hot applications to the chest. She was given iced milk and soda-water with brandy in small quantities at frequent intervals. She was also given the following mixture every four hours: bismuth carbonate, 10 grains; aromatic spirit of ammonia, 15 minims; compound tincture of cardamoms, 10 minims; mucilage, q.s., and water to 1 ounce. On the following day (30th) the vomiting had ceased. The respirations were 66, pulse 102, and temperature 101°. She still had considerable pain over the lower axilla, and a marked friction sound could be heard over this area, and it