purulent discharge, and the child is playing about in bed. He is kept in the hospital, as he is being fitted with a spinal support.

I relate this case, as it shows the rapidity with which irritation follows upon the withdrawal of the drainage-tube and the consequent retention of a small quantity of discharge. It also seems to me to point, as does the preceding case, to the possible good effects produced by the opening and cleansing of the abscess upon the quieting, perhaps even the arresting, of the carious affection of the spine. This point has attracted the attention of other observers.

CASE III. *Perinephritic Abscess from Calculus in Kidney.*—In this instance, the patient suffered from a large chronic abscess about a kidney, in consequence of the formation of a calculus in its pelvis (perinephritic abscess). W. L., aged 56, was admitted into Darken Ward on March 28th, 1876. He had been ailing for five years, his water being reported as at times having been very thick. Lately, he had been obliged to pass urine every hour or two, and had had symptoms which led to his being sounded for stone. The urine had latterly become clearer, but, three weeks ago, he had noticed a swelling in the left loin, where also there arose a great pain. On examination, a large abscess was found in the lumbar region. Pulse, 110; temperature, 102.4 deg.; respirations, 23. On March 31st, the abscess was aspirated, and forty-eight ounces of pus were removed. On April 10th, it was aspirated to twenty ounces; on May 11th, it was aspirated to twenty-one ounces and a half; on June 3rd, it was opened, and thirty-six ounces of pus were let out.

On introducing a probe, it met with septa in various directions; but, by manœuvring, it was at last directed towards the right kidney, and struck against a mass of impacted calculus. The patient was now in a very feeble condition. Pulse, 128; temperature, 101.2 deg.; respirations, 28. He refused food, and sordes were found about the lips and teeth. He was becoming exhausted by the discharge. The only good point about him was, that the urine was better retained, and was clear.

Whilst he was in this state on June 6th, I decided upon endeavouring to distend the abscess-sac, which was effectually done twice; a drainage-tube was inserted and the usual dressings applied. The next morning, the pulse was 92, temperature 99.2 deg., and the respirations natural. The patient felt better; was free from pain, and there was scarcely any discharge. For a few days, a small quantity of limpid fluid exuded, and occasionally the contracting sac was washed with a small quantity of carbolic water. I do not know that this was absolutely needed, but I was anxious to ensure perfect cleanliness. The patient's general condition rapidly improved, and, on June 27th, he was up and about. He was sent home into the country to recruit his health, having been instructed to keep the sinus open, and, when he returns, he will do so, that we may consult as to the propriety of practising an operation for the removal of the calculus from the pelvis of the kidney.

BULBAR PARALYSIS.

By THOMAS S. DOWSE, M.D., F.R.C.P.E.,
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THE disease whose especial clinical features my paper relates is best known in this country as labio-glosso-laryngeal paralysis, but I have adopted the name of bulbar "paralysis"—a name which was first introduced by Wachsmuth. It is much shorter, and directs the attention at once to the seat of the pathological lesion of the nervous centre—namely, the medulla oblongata, from which the facial, trigeminal, glosso-pharyngeal, pneumogastric, and spinal accessory nerves take their origin; and as, at some period or other during the course of the disease, all these nerves are implicated, it is evident that the term "labio-glosso-laryngeal paralysis" conveys a very inadequate idea of the parts involved.

The disease was first noticed by Trousseau in 1841; but it was not until twenty years later that its clinical features were more accurately described, both by himself and by Duchenne of Boulogne. Since then, however, scarcely any disease of the nervous system has been more written about, either directly or indirectly, than paralysis of the parts concerned in expression and speech. To our own countryman Dr. Hughlings Jackson, we are undoubtedly much indebted for the admirable and original work he has done in this as in other branches of nervous disease.

Statistics prove that this malady prevails more among men than women, in the proportion of two to one. It does not carry with it any history of heredity, unless we take into consideration those progressive forms of bulbar paralysis which are by no means infrequently found in

large lunatic asylums, and which are mere continuations of morbid changes in the nerve-substance of the brain and spinal cord.

It has been my experience, and I do not think it will found to be an exceptional one, that paralysis of those parts receiving motor power from nerves originating in the medulla oblongata is extremely rare, unless associated with change in the encephalon or spinal cord. Some few cases have been placed on record where the anterior pyramids, olivary bodies, and lateral tracts were the seats of morbid growths; but these are rare, and can scarcely be said to influence the general question. No one doubts that the medulla might be the seat of a primary interstitial neuritis, resulting in progressive palsy known as glosso-laryngeal paralysis; but this I must hold to be exceptional. I know that there are many men of the highest eminence who maintain that, in its etiology, this form of paralysis is essentially of syphilitic origin; but this does not accord with my own observation, and I am fully persuaded that, in many instances, it is an erroneous supposition. That syphilis is a fertile source of origin for many obscure forms of disease, I have no wish to deny; but I utterly repudiate the fashion which at present prevails, and which ascribes unknown morbid conditions to the effects of syphilis, because these conditions apparently yield after large doses of iodide of potassium have been administered.

I can hardly see how it is possible for syphilitic disease of the membranes, and of the vessels covering the medulla, to produce sufficient lesion of the bulbar roots to give rise to this distinct form of paralysis. The arterial change might advance to the most minute vessels supplying the various nuclei with blood. Then one can understand a lesion of the nerves as a sequence.

I have had a case of basic cerebro-spinal meningitis, where the medulla has been covered with corpuscular lymphoid material, invading its entire surface, yet, during life, there has been no lesion of the bulbar nerves. I have long considered that (as before stated), from its progressive character, the bulbar lesion is of an essentially inflammatory nature; although, in four of my cases, where the bulbar paralysis was most marked, it came on suddenly, associated with paralysis of the extremities.

Again, we know full well that a pseudo-bulbar paralysis can exist, caused by central brain-change, whether this change be the effect of hæmorrhage or of vascular plugging producing mere inanition; and this is more especially the case when such changes originate in the body of the pons Varolii, effecting absolute inhibition of the medullary nerve-centres, and in this case no lesion can be detected after death in the nuclei of origin in the medulla. And this leads us to the consideration of another cause, namely, reflex agency—a rare condition; but I have had one case, which I shall presently detail. I have seen many cases of diphtheritic paralysis, but never one where the tongue has been affected.

Hysterical bulbar paralysis I have never seen, and do not believe in; but I have at present under my care a case, which will be described further on, of amyotrophic lateral sclerosis of the spinal cord and medulla, involving all the cranial nerves, except the first, second, and third. In this case, no power can depress the lower jaw, or make the patient depress it by a conscious voluntary effort. When, however, food is brought to her, the lower jaw falls. I believe it falls by a process as purely automatic as is that of the sucking of the new-born child at the mother's breast.

Thus we find that the causes of bulbar paralysis are numerous, and can be divided into direct and indirect:

DIRECT.

1. Progressive interstitial neuritis
2. Thrombosis of medullary vessels.
3. Hæmorrhage.
4. Morbid growths.
5. Vascular spasm.

INDIRECT.

1. Reflex action from peripheral irritation.
2. Inhibition from shock to central cerebral ganglia.

To all who have considered the physiology of the nervous centres, it must have been apparent that the medulla oblongata constitutes a focus or centre, from which proceed those nerves intimately connected with the phenomena of vitality. Here, in fact, resides (possibly) the seat of sensation (Schroeder van der Kolk). Here, reflex movements pass over to the opposite side; here is the centre of automatic respiratory motions and of deglutition; hence the vagus nerve derives its remarkable influence upon the heart, and an irritated condition of this part produces an excitation of the sexual organs, and influences the secretory action of the kidneys.

And there is abundant proof to show that the vascular supply of the medulla, especially of the corpora olivaria, and the ganglionic cell-groups

which give rise to the bulbar nerves, exceeds that even of the grey cornua of the spinal cord itself. Hence I infer that, in the study of bulbar paralysis, we have a series of phenomena of such high physiological and clinical importance that one can scarcely be wearied in their investigation.

Until the last three or four years, we were accustomed to consider this form of paralysis as progressive and primary; but we now know that it may be stationary, retrogressive, or curable. Dr. Silver was the first physician in this country who drew the attention of the profession to what might be considered the retrogressive form of this disease; but I know of no single instance where a complete cure has been effected, except in the case of a young man, twenty years of age, who has been under my care for the past three years, and in whom all the

Fig. 1.

bulbar nerves were more or less involved. (See Case III.) Before giving the clinical history of this case, which is of great interest, I will draw attention to some of the principal features in the true or progressive type of bulbar paralysis; for although we meet with exceptional cases now and again, it is an almost invariable rule that the disease shall progress from bad to worse in the course of from two to three years. What is the objective sign which first gives us sufficient evidence to state that our patient suffers from a deadly disease, when, to himself or his friends, there appears to be little or nothing the matter with him? And the question arises, whether the first symptoms are really subjective or objective.

Let it be understood, again, that we have to deal with the nuclei of origin of the sixth, seventh, eighth, and ninth cranial nerves, and particularly of the parts which they supply with motion; sensation being rarely affected. Thus some interference of the ocular muscular movements might be evidenced by palsy of the external recti muscles of the eyeballs, but this nerve usually escapes. This is a clinical fact, but anatomically it is unaccountable, for both Stilling and Lockhart Clarke have demonstrated that the sixth and seventh nerves arise from a common nucleus, and, as a rule, where lesion of the sixth and seventh nerves co-exists, the seat of origin is said to be implicated rather than the trunk. Through the seventh, however, and its supply, we have made evident the most objective and primary phenomena; and if we attempt to diagnose disease of the root of this nerve, in the same way that we do some lesion of its trunk (as in ordinary facial paralysis), we shall be mistaken, and led into error.

The first of the facial muscles to become paretic is the orbicularis oris, and the mucous surface of the lower lip becomes exposed. The lip itself is drawn downwards and forwards. We shall see that this, in a later form of the disease, forms a cup, which will be constantly filled with saliva, and it will soon become evident that there is a want of voluntary power to bring the lips together, followed by inability to co-ordinate those movements usually brought into play for kissing and whistling. But before the labial condition arrives at this stage, the movements of the tongue will be interfered with—not the automatic, but the purely voluntary. For instance, the tongue and lips soon become fatigued. If a patient, with incipient glosso-labial paralysis (for in this stage it is confined to the seventh and ninth nerves) be asked to read, his powers of articulation are good enough at first; but in a short time the words are drawled out, and labials and linguals are most imperfectly pronounced, so that, from sheer fatigue, the movements of tongue and lips are rendered temporarily incoordinate. This condition belongs also to other voluntary muscles undergoing incipient atrophic change, where the anterior cornua of grey matter are degenerating. It is a point of clinical and therapeutical interest to which, I think, hardly sufficient attention is usually given.

Thus far we have arrived at the first stage of bulbar paralysis, which may be known as that of fatigue, inducing loss of voluntary power for the due co-ordination of combined muscular movements of the lips and the tongue. The patient may remain in this state for years, as mastication, deglutition, and insalivation are not interfered with. It is when the disease advances beyond this stage that it is most surely and fatally progressive. In the second stage, we find that the ordinary automatic movements of the tongue and lips lose their special characteristics. The patients' attention is constantly directed to the movements of these parts, and they now, for the first time, become conscious that something is wrong. It is this loss of automatic necessitating volitional power which is the sure indicator of central nervous change; the precise and measured character of the movements, the consciousness of the function of volitional activity being excited. With the knowledge that volitional co-ordination is not as formerly under the influence of the will, and the desire to will without the power of will, we shall see shortly, indicate, in the most direct manner, marked central change of the motor centres of the medulla, as distinct from a reflex action induced by lesion of the cortex or of the brain centres. It is now that we find, not the faculty of language, but the power of articulating, to become affected, and this varies according to the muscles or groups of muscles involved. The want of proper pronunciation invariably begins in the tongue. The difficulty of rolling the tongue to give effect to the letter R becomes strikingly manifest, and also of the lips to the utterance of O and U and P, F, B, and M. Then, as the disease advances, the lingual movements cause defective articulation of S, L, R, T, and in due sequence articulation becomes a mere jumble.

CASE I.—The following case, now under my care, will explain the symptoms and signs of the disease in its first stage. J. Maynard, aged 50, was under my care in September 1873. In the winter of 1872, he became for the first time very nervous and agitated, and his limbs would shake under him. When walking across the road he was so fearful that he became transfixed, and could not move a step. In fact, temporarily, he was cataleptic. There were no sensorial defects, giddiness or staggering; no objective signs of disease of cranial nerves; yet, under the least excitement, he seemed to lose all voluntary power. He said that a straw would trip him up, and a puff of wind blow him down. The loss of volition in this case was very interesting. He said that when he tried to do his best, or to exert himself at all, he was unable to do as much as under ordinary circumstances. All the extremities were more or less paretic to motion, but not to sensation. He was dis-

Fig. 2.

charged, and admitted again into the Central London Sick Asylum on July 12th, 1875, in the following condition. He was unable to stand. He said that he could not keep himself in the perpendicular. The first, second, third, fourth, fifth, and sixth cranial nerves were normal. There was defective power in the left arm as compared with the right, and commencing muscular atrophy of the thenar and hypothenar eminences. The under lip had fallen away from the lower row of teeth, and the orbicularis oris was very much weakened. He was able to pronounce some of the labials, but P and F very imperfectly. He had voluntary power to protrude the tongue—in fact, to move it in any direction, unless he became excited or fatigued. Then the lingual movements were at once defective, and heralded the approach of what might be expected.

The best letter-test for noting lingual muscular defect is the articulation of the letter R, and this letter he was unable to pronounce. There were no muscular fibrillations of the tongue (which is a common condition in bulbar paralysis); no impairment of sensation or of the sense of taste; neither could it be said that there was atrophy of this organ; but there was beginning over its surface that trophic change which is so

specific. I allude to almost complete atrophy of the papillæ, combined with a flaccidity and smoothness of the surface, presenting here and there regular furrows. There was some defect in swallowing fluids (not solids), arising from commencing paralysis of the palate. There was also the nasal twang; this, however, was much more marked when the disease was further advanced. (It is this condition which gives rise to the so-called "explosives" of Dr. Duchenne.) As soon as the paralysis of the palate is so advanced that an excessive quantity of air escapes through the nose, the formation of the letters B and P is prevented, because (as Duchenne says) the force of the stream of air which must pass through the mouth and lips for their production is insufficient to overcome the contractile tension of the lips. Here we have evidently medullary disease, which in all probability originated in the anterior pyramids, and is gradually invading the roots of the bulbar nerves.

The notes of this case have been made as brief as possible; and it must be observed that the central lesion was confined to the motor tract, and sensation was in no way impaired. The lesion is essentially progressive, and associated with muscular atrophy of the left upper extremity. In writing of muscular atrophy (and the same condition pertains to bulbar atrophy), Charcot supposes that the degenerative processes tend to creep among the ganglion-cells of the anterior cornua, so as to destroy some cells earlier, some later, some more and some less completely; while, at the same time, other individual cells and whole groups of cells remain unaffected by the disease. In the case just narrated, it will be seen that the lips and pharynx were paralysed before the tongue, and Dr. Duchenne has recorded several such cases.

We now come to the consideration of the more advanced stages of the disease. We have seen that the nuclei of the facial, or the nuclei of the hypoglossal, or even that of the spinal accessory or pneumogastric, may be primarily involved. In this order of sequence we see: 1. That the facial expression is altered; 2. That the lingual movements are interfered with; 3. That the articulation of certain letters is rendered impossible, until the only sound which the patient can utter is purely laryngeal; and, finally, when the laryngeal muscles themselves are palsied, the only sound coming from the larynx is a sort of grunt. It is now that other troubles become manifest. The third division of the fifth nerve not unfrequently is influenced, so that the movements of the lower jaw in mastication cannot be duly performed; and in some of my cases this has been a marked feature. I believe that, in the majority of instances where the medulla has been examined, no lesion of this nerve has been discovered, so that we are willing to admit that the part which it plays in bulbar paralysis is rather reflex than direct. Schroeder Van der Kolk, in speaking of this nerve, says: It is quite unnecessary to dwell upon the use of the connections of the trigeminus, considered as a reflex nerve, with the glosso-pharyngeal, vagus, accessory, and hypoglossal nerves in swallowing, inspiration, coughing, and sneezing. Hitherto, as far as the facial is concerned, we have seen that the orbicularis oris is the only one of the facial muscles involved in the first stage of this paralysis; and, as I have before stated, it is a point of great physiological importance. In what is commonly called facial paralysis, we know that invariably the trunk of the portia dura is affected, in all probability, before its entrance into the aqueductus Fallopii, and we have complete paralysis of all the facial muscles of one side. Yet, in bulbar paralysis, we find the converse of this, when its root is primarily involved, and (presumably) only some of its nuclei of origin. Romberg says: "A peculiar feature in the central paralysis of the facial nerve in disorganisation of the brain is that, with few exceptions, the entire distribution is not affected; but those fibres only are implicated which supply the muscles of *alea nasi* and the upper lip, and are the agents of the respiratory functions of the facial." Thus it seems to be diagnostic, as we find in primary bulbar paralysis that the orbicularis oris is first involved, and is diagnostic of central rather than of peripheral change; and, strange to say, this is made still more evident in hemiplegia, where we have a slight facial palsy on the side of the paralysis, arising from some of the superficial fibres, as they pass through the pons Variolii, being involved. From the investigations of Lockhart Clarke, we know that the facial nerve arises from a common nucleus with sixth; hence there ought to be some palsy of this nerve, if there be a lesion of the facial at its origin, but clinically this is not found to be the case. This, however, is a clinical fact: that the root of the facial shall be the seat of lesion when its peripheral supply to the orbicularis oris has been cut off, not unilaterally, but bilaterally. As the disease advances, the facial paralysis increases, so that the buccinators become involved, and a mask-like appearance of the face supervenes. Up to this period, although the movements of the tongue have been gradually more and more limited, yet its power of protrusion has not been annihilated. But now we see it lying immovable behind the lower row of teeth; saliva is constantly dribbling over the depressed lower lip;

mastication is very imperfectly performed; and the muscles of the palate, pharynx, and larynx are involved.

The following brief clinical notes of this stage of bulbar paralysis are typical.

CASE II.—J. S., aged 28, was in the army for some years, and served in Canada, where he had a sunstroke. For this reason, he was discharged from the army, and returned to England. In the summer of 1873, during a very hot day, he was brought home in a fit, and, when consciousness returned, he stuttered and had difficulty in bringing out his words. For more than a month, he frequently complained of great pain in the back of the head. In the following January, he was seized with another fit and partially lost consciousness, when he was admitted into the Central London Sick Asylum with well-marked bulbar paralysis. I find the notes of his condition in my case-book to be as follows.

The lower jaw could not be depressed for more than half an inch; the tongue was immovable; saliva was dribbling from the mouth, and

Fig. 3.

there was considerable paralysis of the muscles of each fore-arm, with deficiency of grasping and co-ordinating power—more marked, however, in the left than in the right. He walked slowly, but did not stagger or appear to be giddy. There was a calm and slightly idiotic expression of countenance, and, upon examining the head, a deep furrow of depressed bone was found in the direction of the left parietal-lambdoidal suture. When restrained to be fed by means of the stomach-pump, he was sometimes excited, and became rather violent; but this state was only momentary, and passed off with the cause. After he had been in the hospital about a week, the use of the stomach-pump was discontinued, and fluid nutriment was passed into the stomach through the nose by means of India-rubber tubing. Upon more than one occasion he was found partially unconscious, in a state almost amounting to asphyxia, which arose from the pressure of a bolus of food upon the epiglottis. When in this condition, the sphincters were relaxed, and faeces and urine passed involuntarily. Notwithstanding this, there was at times a doubt as to the extent of voluntary power which he possessed over the paralysed parts. One day, when the tube of the stomach-pump was being introduced, some difficulty was experienced when it came to the pharynx, and one of the attendants gave him pain by pressing too hard upon his leg. This drew his attention away, and, not only did the tube at once pass easily, but his jaw dropped fully.

The parts paralysed, the amount of palsy, and the manner in which these conditions present themselves, will be best considered in the following order.

First, then, with regard to facial expression: it cannot be said that this case presents marked labial paralysis over that of several other muscles of expression. There is considerable paralysis, however, about the orbicularis oris, which prevents him from bringing the lips together, as in whistling, pronouncing some of the vowels, or expiring a column of air; and, from the lips being at all times slightly apart, there is constant dribbling of saliva from the mouth. When made to laugh, as a rule, those muscles which draw up the angles of the mouth—the zygomatici and risorii—usually act incoordinately, giving one at first sight the impression that the muscles on one side of the face are considerably more palsied than on the other. This is true, in a measure, and presents a point of interest. Invariably, when this act is performed, the left angle of the mouth is drawn up before the right. The lower jaw is partially depressed, and the patient has no voluntary power to extend it beyond a given point; yet, when he performs an extra inspiratory effort, as that associated with yawning, when the shoulders are raised and the chest and head become fixed, the depression of the lower jaw—namely, the digastric, the stylo-hyoid, and the genio-hyoid—appear to come into play; the hyoid bone becomes elevated, and the jaw

drops, and returns to its former position by an apparently natural and voluntary effort. This appears to be the chief complication which makes this case to differ from other recorded cases of glosso-laryngeal paralysis. The tongue is almost motionless. When asked to protrude it, he can at times (this is not invariably the case) move it just over the edge of the lower row of teeth, and, when drawn out forcibly by a pair of forceps, it is smartly retracted—showing that the genio-hyo-glossus muscle is not so much palsied as the hyo-glossi and linguales, as he is totally unable to render it concave, or move it from side to side. The nutrition and sensation of the mucous surface of the tongue does not appear to be interfered with. There was some difficulty in examining carefully the velum and pillars of the fauces and the interior of the larynx, from his not being able to depress the lower jaw sufficiently. After as exact an observation as possible, the isthmus faucium, including the palato-glossi, palato-pharyngei, uvula, as well as the levatores and tensores palati, seem almost devoid of all tactile sensibility. On stimulating these parts by means of a quill, the muscles do not respond, the uvula and velum pendulum palati remaining in their flaccid condition, and there is no reflex movement of the middle and inferior constrictors to produce the feeling of nausea which invariably accompanies this act; but, directly the posterior wall of the superior constrictor muscle is touched, it gives him pain. Trousseau says that, in these cases, sensibility was everywhere normal, and that irritation of the mucous membrane of the soft palate produces motion of the velum by reflex action. The patient is almost devoid of masticating power, not so much from absolute inability to raise and depress the lower jaw as from the absence of alternation of lateral movements, which are influenced by the external pterygoid muscles. He had scarcely any power to exercise the first or second stages of deglutition. This is readily accounted for by the almost perfect immobility of the tongue. It cannot be pressed upward against the hard palate; and its base cannot be retracted at the same time that the larynx and pharynx are raised. The epiglottis, which in the second stage would naturally fall upon and close the upper aperture of the larynx, remains partially inactive, and does not allow the food to pass over it to be grasped by the pharyngeal constrictor. Hence arises the condition of partial asphyxia, from which he had not unfrequently suffered. The method he adopts to take solid food is this: he crams the mouth until it can hold no more; and after the mass of food has become softened by saliva, he pushes it with his finger through the isthmus of the fauces over the epiglottis, where it falls by gravitation into the grasp of the inferior constrictor of the pharynx. When once here, this muscle acts to propel it onward. From this, it was concluded that the inferior constrictor does not participate to such a degree in the paralysis—at all events, in the present stage of the disease. This might perhaps be accounted for on the ground that this muscle receives a branch from the external laryngeal nerve, in addition to the supply from the pharyngeal plexus. It sometimes happens that the fluids which he is attempting to swallow are forcibly ejected, not only from the mouth, but through the nostrils. This is the result of some portion of the fluid finding its way through the chink of the glottis, in consequence of the incomplete closure of the aryteno-epiglottidean folds. The next point to be noticed, and a most important one, is that the man is absolutely dumb. He cannot pronounce a consonant or vowel, and the only sound which emanates from the larynx is a grunt. This complete aphonia, according to Dumenil, depends on two causes—paralysis of the muscles of the larynx, and paralysis of the thoracic muscles. Indeed, physiological experiments prove that, when the spinal accessory is torn off at its root, complete aphonia ensues from relaxation of the vocal cords.

In this case we have a complete delineation of bulbar paralysis, in its latest stage. It is not progressive, but *stationary*. The man is now in good health, but he has complete paralysis of the bulbar nerves. From the sudden nature of the attack, one is led to suppose that its causation was due to hæmorrhage into the medulla and superficial fibres of the pons Varolii—bulbar paralysis.

We have to consider, in reference to this man's present condition, one or two points of interest which have not yet been alluded to, namely, the increased salivary secretion, the aphonia, dysphagia, and dyspnoea. The overflow of saliva commences at the same time that the first stage of deglutition becomes involved; and is due most probably to this fact, that the patient has little power to swallow it. There can be no doubt that its secretion is far beyond the normal quantity, and resembles a condition of ptyalism. The cause of this must be referred to paralysis of the chorda tympani nerve. The difficulty of swallowing can be clearly traced through each of its stages. The first stage is simultaneous with palsy of the ninth nerve; the second, with paralysis of the motor branches of the glosso-pharyngeal; and the third, with paralysis of the pneumogastric, through its motor influence derived from the spinal accessory. When the nuclei of the pneumogastric and

spinal accessory are invaded, we find almost total inability to pass solid food into the œsophagus. Fortunately, however, for the patient, although the palato-glossi and palato-pharyngei, with the intrinsic muscles of the larynx, are almost devoid of mobility, their sensibility is to a much less degree implicated; and so by reflex movements, however inco-ordinate the act of deglutition may be performed, although in the effort patients are often barely saved from suffocation.

In one of my cases, paralysis of the vagus was most marked, and caused very distressing symptoms. The inspirations were deep and prolonged, and gave to one the idea that the chest alone, and not the lungs, was expanded. At times, during these attacks, temporary breathing was prolonged almost to complete asphyxia, and artificial respiration had to be resorted to. The pulse would be scarcely appreciable at the wrist, and the sphincters were invariably relaxed. It must be remembered, though, that these paralytics, as a rule, retain voluntary power over the sphincters up to the time of death.

Thus far we have presented to us all the progressive and characteristic signs of bulbar paralysis. As I have shown in Case II, it does not kill for some time, if the patient be carefully and regularly fed. This is a point of the highest importance in treatment; and will more than anything else retard the fatal issue, which, however, under any circumstances, is merely a question of time; for as enervation increases, there is marked failing of power all over the body—the upper extremities are feebly raised to the mouth to wipe away the increased flow of saliva, and the lower limbs are dragged along until strength is lost—for this is the last display of voluntary power. The reclining posture is impossible, on account of the paroxysms of suffocation; and death often arises during sleep, from the flow of saliva into the larynx, or from plugging of the bronchial tubes with viscid mucous, which the patient is unable to expel.

[To be continued.]

ILLUSTRATIONS OF PRACTICE IN COUNTRY CORONERS' COURTS.*

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THE personal anecdotes of which this paper in great measure consists were read by me (with one or two exceptions) to the Bath and Bristol Branch of our Association some years ago, and would not have been reproduced here had it not become evident, from speeches in Parliament and articles in professional as well as general periodicals, that public attention has now, however tardily, been directed to this matter, and that the juncture is, therefore, one in which every man who believes he can say anything to show the faultiness of the existing system, and help towards its improvement, is bound to speak. And I do not think the audience before whom I have the honour to stand is inappropriately appealed to on this subject; for not only is our profession fitted, by its position and intelligence, to take its place amongst the foremost in all matters of social well-being, but we have a special interest in this matter. The position of a medical witness in a coroner's court is one not only of heavy responsibility as regards others, but of the utmost importance to his own professional reputation; and, as it is a position which either of us may at any moment be called upon to take, we may well concern ourselves to know something of the tribunal whose deliberations we have to assist, and to use our influence in adding to its efficiency and impartiality. In our days, science is too often hired to be the handmaid of crime, and the pair together sometimes weave a veil through which the best directed inquiries fail to penetrate. There is also reason to fear that some crimes, such as infanticide, are increasing in frequency as well as in subtlety amongst us. It, therefore, behoves us to see that our sentries are wide awake with arms and accoutrements all in good order. That such is not the case, that the safeguard against crimewhich society is supposed to derive from coroners' inquisitions exists more in theory than in practice, has long been well known to medical men, especially those practising in country districts; and several cases of more or less notoriety have occurred in the last two or three years, which have at last awakened the interest of statesmen and the public. In order, as far as in me lies, to keep this interest awake in your minds, I bring the following personal experiences before you, and feel sure that you will admit that such a list, drawn from the knowledge of one man moving in no very extended sphere, reveals an amount of indecorum in the coroner's court and incompetency in its chief officer, which shows the urgent need of alteration and improvement. My first instance is as follows.

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