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SOME CLINICAL ASPECTS OF VOCAL CORD INACTION

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" *Time and Experience alter all perspectives* " (HENRY ADAMS).

FOREWORD

IF the truth embodied in this epigram were not so fundamentally applicable to all human activities, there would be little reason for returning to a subject which was the basis of two previous communications, viz. " Some clinical observations on paralysis of the left Vocal Cord " (*Journ. of Laryng. and Otol.*, June, 1918) and " Mechanical Immobilization of the Vocal Cords " (*Brit. Med. Journ.*, October 15th, 1927).

But during the decennium which has elapsed since the latter contribution, further experiences have so far altered the perspective of my views therein contained as to call for some focal readjustment if they are to be again envisaged in the light of knowledge since acquired. As before, their exposition will represent an attempt to find some explanation of those not infrequent cases in which hoarseness, or some less definite alteration of the voice, has been found to be due to an inactive, but otherwise normal vocal cord, which could not be traced to a comparatively gross lesion involving the origin, course, or distribution of the corresponding recurrent laryngeal nerve.

Herbert Tilley

In order to save readers the trouble of referring to those earlier publications, the case histories they contained will, when necessary, be included again together with others which, with one exception, have not hitherto been recorded. Furthermore, each of them will immediately be followed by comments which reflect the current opinions of the writer. For this reason it would be idle to imagine that they will entirely coincide with those held by other laryngologists who have had equal opportunities for drawing their own conclusions. If these betray considerable divergences from mine, it will be clear evidence that the chosen subject presents problems for the solution of which " Judgment is difficult and Experience fallacious "

CASE I.—*Acute Oedematous Laryngitis.* (Seq. Permanent bilateral crico-arytenoid ankylosis with adducted cords.)

Between 1893 and 1894, a man of middle age was hurried one evening into a Throat Hospital where I was house surgeon. The severe degree of inspiratory stridor, the dusky appearance of his face bedewed with drops of cold perspiration, and a restless anxiety, were such obvious signs of impending suffocation that, without any mirror examination of the larynx, we placed the patient on an out-patient couch, and I did a low tracheotomy. General anæsthesia was out of the question, and methods of local infiltration analgesia had yet to be discovered.

The following day, laryngoscopy revealed such an intense œdema of the aryepiglottic and laryngeal mucous membranes, that only fleeting glimpses could be obtained of the middle thirds of the closely adducted and motionless cords. The pulse was 84 and the temperature 101·6. No local or distal complications hindered a rapid general convalescence. Meanwhile, the laryngeal œdema gradually subsided but the vocal cords showed no signs of returning activity. The man lived in London and, for at least twenty-five years, it was possible to make periodical inspections of his larynx. He could never dispense with the cannula because the cords were permanently stabilized near the mid-glottic line by ankylosis of their respective crico-arytenoid articulation. Each of these cartilaginous structures remained enlarged owing to the initial perichondritis.

Comments.—It is very doubtful whether any bacteriological examination was made of the patient's throat or laryngeal secretions at the time, but it would be safe to infer that the clinical manifestations would to-day be

regarded as typical of an acute hæmolytic streptococcal laryngitis.

" Early impressions are the most lasting ", and from that clinical experience I received two :—

(1) The comparatively small handicap imposed on the patient's daily activities (manager of a coffee stall) when he had become accustomed to wearing his cannula. This important and practical detail has frequently been emphasized, and occasionally demonstrated on some of his patients, by Sir St.Clair Thomson, who has laid down the general principle that, given the choice of an operation for providing a permanently free air-way in cases of chronic non-malignant laryngeal obstruction, there can be no comparison between the advantages of a low tracheotomy as contrasted with those which might be claimed for other surgical procedures, e.g. progressive dilatation of a fibrous laryngeal stricture by intubation. After a recent conversation with him on this subject, he kindly sent me the following summary of his views. (They are the more acceptable on this occasion because, as will be seen later, there are conditions in which I maintain that laryngotomy has particular advantages over the lower operation.)

Sir St.Clair writes : " My thesis is that in many cases of established glottic stenosis, the various methods recommended for re-establishing the normal air-way do *not* succeed in satisfying normal air-hunger and, not infrequently, leave permanent damage to the voice. Secondly, I claim to have proved that the prejudices against wearing a permanent tracheotomy tube have no foundation in fact. I can only allow that there is some slight social disadvantage, much less noticeable in the female whose dress allows of her concealing the tracheotomy tube than in the male. The other disadvantage is the very remote risk of drowning if the patient were suddenly plunged into deep water. So far as general health is concerned we find that, so full is nature of compensations, there are no drawbacks from the loss of nasal respiration. There is no increased tendency to bronchitis ; head colds are even less frequent than in normal breathers ; the wearers of the permanent tube can play tennis or golf, ride, skate or dance with as much vigour and enjoyment as the normal being. Women can bear children or work in factories and gain their livelihood without suffering any handicap.

" Particularly in the cases you mention (bi-lateral adduction

of the cords), where there is no hope of restoring a mobile glottis, it is ' the only way.' "*

(2) It was equally surprising to find that a vocal cord could be immobilized by ankylosis of the corresponding crico-arytenoid articulation. As already implied, that knowledge was acquired within two years of leaving my *alma-mater* where we, as clinical undergraduates, had been taught to regard an inactive cord as one of possible signs of a lesion—generally of a grave nature—involving the homolateral recurrent laryngeal nerve somewhere between its central origin and final distribution to the adductor and abductor muscles of the cord. But, so far as my recollection goes, no mention was made of a local mechanical factor which could effect a similar immobilization. This omission seemed the more strange when, subsequently, I found that twelve years previously, Dr. (later Sir) Felix Semon delivered five lectures at the Medico-Chirurgical Society of London, entitled " Mechanical Impairment of the Crico-arytenoid Articulations " in which all aspects of the subject were fully considered.

Incidentally, he laid particular stress on the serious issues which might follow a wrong diagnosis as between true paralysis of a cord and its fixation by the articular lesion (vide *Medical Times and Gazette*, 1880).

It would far transcend the scope and purpose of this contribution to explain the silence of our mentors on a matter of such clinical significance. It must, therefore, suffice to mention three conditions which, at the time, hindered its transmission from teachers to students :—

(1) Knowledge which implied concentrated study of the diseases of any particular organ, or expert methods of treating them, stirred to their roots the conservative instincts of most leaders of the profession. Such attainments implied " specialism " which, in the opinion of a contemporary President of the Royal College of Physicians, was " the Canker of Medicine ".

(2) In the majority of the London Schools of Medicine, the Throat Department was placed in charge of an assistant general physician or surgeon, and it was not compulsory for students to attend such special clinics. Consequently, many

* " The Permanent Tracheotomy Tube ", *Lancet*, June 11th, 1927; " Permanent Tracheostomy in Stenosis of the Larynx ", *Clinical Journal*, November 25th, 1931.

of them qualified without ever having seen the vocal cords in a laryngeal mirror.

(3) The time-lag between the discovery of a scientific fact and its general acceptance by the medical profession. In his day, Sir Hughlings Jackson computed the interval to be about twenty-five years! Furthermore, in the early 'nineties X-rays and the blood serum test for syphilis still awaited their respective discoverers—Roentgen and Wassermann.

If these adverse factors be kept in mind, no blame can be attached to those who taught us " the truth, nothing but the truth " but not " the whole truth ".

CASE II.—*Acute Laryngitis.* (Seq. Temporary inaction of the left vocal cord.)

In 1895 a medical practitioner was laid up with symptoms which so closely resembled those of acute rheumatic fever that the treatment accorded him had been based on that diagnosis. The illness began with a sore throat, pain and swelling of some of the larger joints. Temperature 101·2. These symptoms were quickly followed by pain in the left side of the larynx which was much aggravated by swallowing. Mirror examination reflected general laryngitis with pronounced œdema of the left crico-arytenoid mucous membrane and the relevant cord immobile in the position of adduction.

With rest in bed and anti-rheumatic treatment, convalescence was, in due course, established, the voice returned and the laryngeal pain subsided.

About six weeks later, I re-examined the larynx, but could find no traces of the previously observed lesions.

Comments.—It would be beyond my capability to discuss whether " Acute Rheumatic Fever " was the correct diagnosis of this case because, so far as current literature may be regarded as authoritative, there does not appear to be a concensus of opinion among pathologists and bacteriologists with regard to the ætiological factors of that disease. But the experience was valuable in that the laryngeal reaction to infection was almost entirely confined to the *left* cord and articulation, although both of the latter regained their normal mobility. This temporary failure should be kept in mind when reading Case III, because the main object of this communication will be to show that, other things being equal, the left vocal cord and articulation appear to be more vulnerable than the right. For many years afterwards, it was often a matter of surmise as to

what would have been my diagnosis and prognosis had the patient's left cord remained inactive and I had seen it thus for the first time. Presuming the absence of any physical signs in the chest, or evidences of a neurological lesion, there can be little doubt that the first answer would have been " paralysis of unknown origin ". The prognosis? A severe restriction of any of the patient's daily activities which demanded excessive physical exertion and, what might have proved even more difficult to bear, the realization of a sword of Damocles for ever over his head.

CASE III.—*Acute Hæmolytic Streptococcal Laryngitis.* (Seq. Permanent left crico-arytenoid ankylosis and cord immobilization.)

Early in February, 1935, a medical confrère was nearing convalescence from a major abdominal operation when symptoms of mild toxæmia developed which were quickly followed by hoarseness, pain in the lower pharynx and mid-œsophageal region. The latter symptom was much intensified by swallowing. In addition, there was a dry, persistent and irritating cough which gave the patient little, or no, rest at night. For present purposes, it is only necessary to describe the chief local conditions as I saw them a few days after their onset. They were, a pronounced anæmia of the soft palate and faucial regions, œdema of the aryepiglottic and supra-glottic mucous membranes, below which only the middle thirds of the inactive cords were visible, and sufficiently adducted as to cause slight inspiratory stridor. Unfortunately, this symptom increased and soon reached a stage at which *laryngotomy* became a matter for serious consideration. In the meantime my instruments had been sterilized and all arrangements made for the operation at a moment's notice. But, as so frequently happens in surgery, " preparation cancels necessity ", and so it was in this instance because, just about the time when further delay seemed unwise, the stridor became less and within a few hours the danger point had been passed.

Subsequent mirror examinations revealed progressive resolution of the œdema and definitely increasing movement of the right vocal cord, but none could be detected in the left. In due course the former regained its functions, while the opposite one remained fixed, slightly abducted, with its respective arytenoid cartilage tilted slightly downwards and forwards towards the mid-glottic line.

Unhappily, the constitutional symptoms of streptococcal toxæmia persisted and, in the following November, the patient succumbed to myocardial failure.

Comments.—While the early laryngeal symptoms caused considerable discomfort which was followed by the mental

anxiety of increasing stridor, his most persistent complaint was the mid-œsophageal pain which was acutely exaggerated even when warm liquid nourishment was swallowed. His own diagnosis of " ulceration " was probably correct because, on one occasion, I saw a large clot of blood in the left pyriform sinus.

What impressed me most at the first examination was the non-œdematous, wax-candle pallor of the soft palate, fauces and oro-pharynx. Perhaps this anæmia was rightly regarded as an evil omen of that lack of resistance to infection which became so evident at a later period. During the earlier weeks of convalescence, we combined to write a short article on " Acute Streptococcal Laryngitis " as exemplified by his recent personal experience of the symptoms, and the clinical conditions which I was able to observe. At the same time, the opportunity was seized for a wider review of the subject (*Brit. Med. Journ.*, 1935, xi, 3).

Although our knowledge of acute streptococcal laryngitis has become more diffused within recent years as a result of such outbreaks of " epidemic sore throat " as have occurred in Bournemouth, Brighton, Chelmsford and Leigh (Lancs.), sporadic examples must be rare, otherwise forty years would not have passed between the second and third of the above case records, although in the meantime, hospital clinics alone have provided me with ample chances of meeting similar cases had they only been of average frequency. To be prepared for such eventualities, the younger generation of laryngologists would be well advised to read " Acute Streptococcal Infections of the Throat " by E. D. Davis (*Brit. Med. Journ.*, September 26th, 1936, II) and " Acute Tonsillitis ", an account of some recent Streptococcal Infections by F. C. Capps (*Brit. Med. Journ.*, May 25th, 1935, i, 1061).

CASE IV.—*Acute Streptococcal Tonsillitis.* (Seq. Oedematous Laryngitis. Laryngotomy.)

In June, 1935, a man, aged 72, had been laid up for five days with acute tonsillitis, followed by increasing laryngeal obstruction. When I examined him, the constitutional symptoms and laryngeal conditions were so like those in Case I as to need no repetition. The only additional feature was an obvious swelling of the lower pole of the right tonsil, which was incised without delay and thereby gave free exit for about two teaspoonfuls of pus. Cultures from this gave an almost pure growth of a hæmolytic streptococcus.

Mirror examination revealed an intense and turban-shaped œdema of the epiglottis and aryepiglottic folds. In the upper central region of the obstruction there was a small dark aperture through which it seemed incredible that sufficient air to maintain life could be inspired. Having sprayed this area with a 1/1,000 adrenaline solution, I advised a laryngotomy with as little delay as possible. The operation was performed within two hours by the patient's medical adviser, and was followed by an uninterrupted convalescence. When seen last year (1937) the only laryngeal defect was a slight loss of tension in the (active) *left* vocal cord.

In view of the paragraph which preceded Sir St. Clair Thomson's references to tracheotomy—with the general purport of which there can be little, if any, dissent—the case history just recorded affords an opportunity for explaining why laryngotomy was advised in the last two cases, although it only became necessary in the second. The two reasons for advising that operation were as follows :—

1. *Anatomical.* It may be taken for granted that œdema cannot develop without the presence of, more or less, loose connective tissue. This axiom could not be better exemplified than by the accompanying illustration of a transverse section of a vocal cord prepared by the late Prof. S. G. Shattock, for the loan of which I am indebted to Dr. Irwin Moore (vide *Journ. of Laryng., Rhin. and Otol.*, March, 1920). It clearly demonstrates that from its outer ventricular limit to the central edge and downwards over the subglottic area, the surface is covered with squamous epithelium based on elastic tissue which is thickest at the junction of the upper and median surfaces of the cord. " Inferiorly the elastic tissue passes into the thin expansion of the lateral expansion of the crico-thyroid membrane which is fixed below to the inner edge of the upper border of the cricoid cartilage."

Consequently supra-glottic œdema is limited to the outer or ventricular border of the cord and therefore a laryngotomy tube would be well below the superjacent obstruction.

2. From the clinical aspect, it must be remembered that by the time a streptococcal œdema demands surgical relief, a considerable toll has generally been made on the patient's constitutional resistance. Therefore, with a choice of operations having the same end in view, the one should be selected which can be performed in a few seconds, is practically bloodless, causes little, if any, mental or physical shock, and leaves

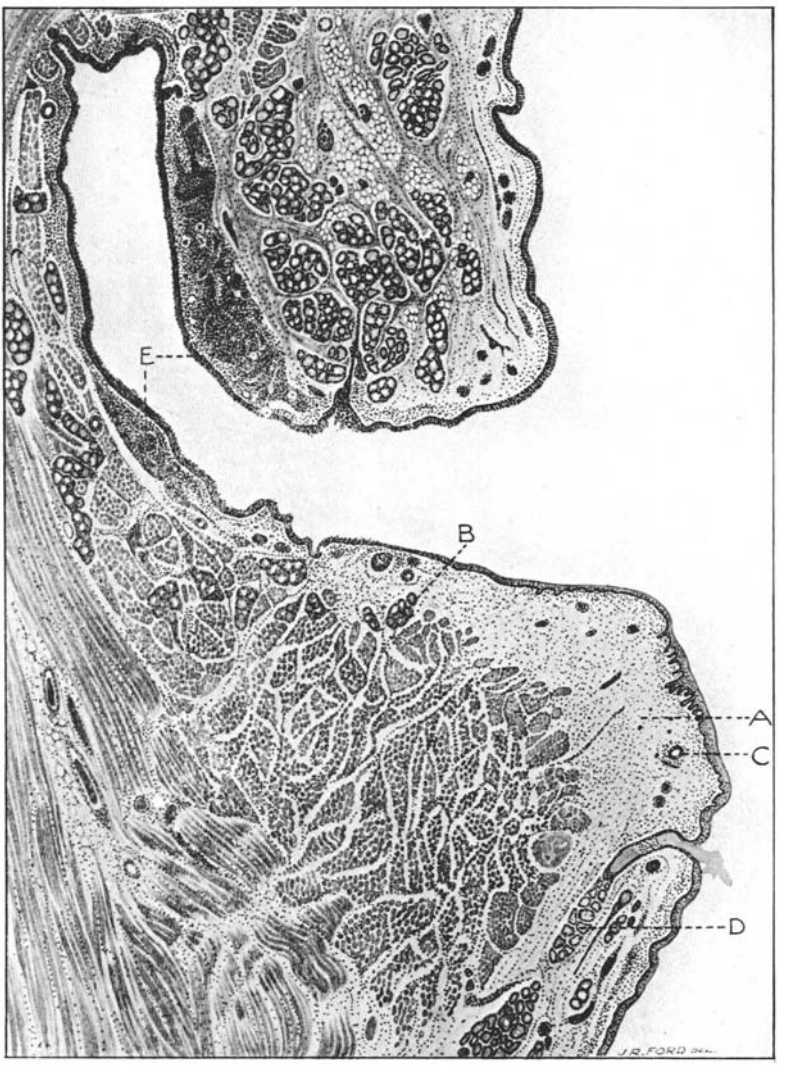

Fig. 1.

Coronal section through middle of normal human (female) larynx, showing the distribution of the gland tissue and the lymphoid tissue of the ventricle (logwood and eosin stain; 2-in. obj.). A. Elastic tissue of cord. B. Gland acini lying in periphery of the cord. C. Gland duct, near surface of summit of cord. D. Gland acini lying in elastic tissue and in an area covered with squamous epithelium. E. Lymphoid tissue in wall of ventricle.

(*Preparation by Prof. S. G. Shattock, F.R.S., specially drawn for Dr. Irwin Moore.*)

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an almost invisible scar or dimple which, in the case of women patients, may be a matter of supreme importance.

Note.—In direct numerical sequence, there will now follow very brief case histories of fourteen patients in whom, with one exception, the left cord was paralysed some weeks or months before the patient consulted me for the consequent " chronic hoarseness ". The deductions drawn from them will be included in one general " Comment " followed by a " Conclusion ".

CASE V.—An adult, aged 52, had been hoarse for three months. His *left* cord was motionless on phonation. The patient was an Austrian and had consulted physicians in London and in his own country, a German laryngologist of European reputation, and an expert radiologist who had provided him with excellent skiagrams of his chest. None of these investigators had detected the cause of the laryngeal defect and, as I anticipated, my first (and only) mirror inspection did no more than confirm what had already been observed. But five years later I saw him engaged in a golf competition and, therefore, it would be fairly safe to presume that whatever may have been the factor of the cord paralysis, it is unlikely that it was of a grave nature.

CASE VI.—Male, aged 47. Hoarse for two months. Mirror examination reflected an inactive and adducted *left* vocal cord. The patient also complained of breathlessness on slight exertion, discomfort in heart region and frequent pain in *left* arm. Diagnosis: Probably aortic aneurysm. This was confirmed by a consulting physician and radiologist. Three months later the intrathoracic lesion proved fatal.

CASE VII.—Male, aged 32, died from acute pneumonia of the left lung three years after he consulted me for hoarseness which was accounted for by *left* cord paralysis. But two years after his decease, his daughter informed me that, in the meantime, his voice had completely recovered.

CASE VIII.—A particularly healthy looking young woman, aged 29, had been hoarse for three weeks following a bad cold in the head. The *left* cord was stabilized in the position of adduction. Three months later severe pain developed in her left ankle and heel. Rest in bed and the ordinary medicinal treatment of gout having failed, she took a course of the Bath mineral waters. After a short while the pain disappeared and the voice gradually recovered its normal resonance. On her return to London I could find no trace of the previous laryngeal defect.

CASE IX.—Male, aged 30, became hoarse while under treatment for left pulmonary tuberculosis. Mirror examination revealed an inactive, adducted, but otherwise normal *left* vocal cord. There were no signs suggestive of tuberculous laryngitis. Three months later the patient died. It is, of course, possible that the cord was truly paralysed by involvement of its recurrent laryngeal nerve in a pleuritic adhesion or, perhaps, subjected to the pressure of an enlarged mediastinal gland.

CASE X.—Male, aged 53. Loss of voice for two months following a touch of influenza. *Left* cord immobolized and adducted. The patient wrote to say that his voice returned two or three months after my examination of his larynx.

CASE XI.—A married woman, aged 36, had been hoarse for a fortnight. The *left* vocal cord was inactive and adducted. Her consulting physician could find no physical signs of a chest lesion. The vocal disability persisted for several months, but when re-examined about a year after its onset, the cord had regained its normal mobility and the voice its natural resonance.

CASE XII.—A male, aged 49, solicitor, consulted me for loss of voice for three weeks. The *left* vocal cord was immobilized in the position of adduction. There were no physical signs in chest, and no evidence of any central nerve lesion. When re-examined eleven months later, the cord activity was normal. He has no recurrence of the hoarseness and is still at work (1938).

CASE XIII.—Male, aged 67, a military officer, had been hoarse for more than a year. It came on immediately after an unusual amount of shouting on parade during a heavy gale. *Left* cord inactive and adducted. In answer to a follow up enquiry, he reported that by the end of four months after his visit he could talk or sing as well as ever, and added, altogether I was hoarse for eighteen months.

CASE XIV.—Male, aged 67, with more or less chronic hoarseness and a clear history of syphilis. The *left* cord was motionless and closely adducted. The relevant crico-arytenoid articulation was (perhaps) slightly enlarged. Both cords were rather congested, their edges ill-defined and on the upper surfaces of each were a few small patches of leucoplakia. Much larger plaques covered the dorsal surface of the tongue. After six months of appropriate treatment, the left cord had regained its activity and, with the exception of a mild congestion, normal conditions prevailed and the patient had a clear voice.

CASE XV.—During general convalescence from faucial diphtheria a child, aged 5, began to show some difficulty in swallowing. Her

voice was hoarse and devoid of resonance. On examination, I found complete palatal paralysis and the *left* vocal cord inactive and adducted. When re-examined a month later, these symptoms and signs had disappeared.

CASE XVI.—Female, aged 42, developed a husky voice after acute tonsillitis. Examination showed the *left* vocal cord motionless and slightly adducted. A later inspection showed that normal conditions had been restored. (Probably a case of diphtheria.)

CASE XVII.—A surgeon gave precisely the same history, and inspection of his larynx showed a similar *left* cord defect. But in this instance, the patellar reflexes were practically absent and there was a considerable loss of accommodation. There can be no doubt that the primary tonsillitis was a Klebs-Loeffler infection.

CASE XVIII.—This, the last case, affords a clinical history which is unique in my experience.

R.B., aged 18, whose build might be envied by any young athlete, was sent to me by a colleague with the following history. " His voice was clear when going to bed on September 15th, 1936, but was quite hoarse the next morning. No preceding general, nasal, nor throat symptoms. I saw him a few days later and found the *right* vocal cord slightly adducted and inactive on phonation. No history of any constitutional infection, nor could I detect any physical signs in his chest. Neurological examination also negative." Mirror examination added nothing to my friend's observations, except (possibly) the patient's volunteered statement that if he turned his head well towards the right shoulder the hoarseness was much worse. On March 3rd—a little more than five months after the onset of hoarseness—he awakened to find a complete restoration of his voice.

A subsequent report from my confrère, stated that the cord had regained its natural functions.

Comments.—The most casual reader of these eighteen clinical records could scarcely have failed to notice that, when both cords and/or their corresponding crico-arytenoid articulation were involved the *left* structures proved to be more vulnerable than the right. This hypersensitivity may be more clearly demonstrated by the following extracts taken from the case histories.

1. In a mild type of acute laryngeal infection, the œdematous reaction was limited to the *left* vocal mechanisms (Case II).

2. An acute hæmolytic streptococcal laryngitis caused an equal degree of bilateral œdema which stabilized both articulations. The right recovered its functions, while the *left* was permanently ankylosed (Case III).

3. A severe vocal strain (shouting) rendered the *left* cord inactive for eighteen months, although the right one was in no way affected.

4. As with other nerves, those which innervate the vocal cords may be paralysed by blood-borne bacterial toxins and/or chemical poisons. Cases IX, X, XIII, XIV and XV were examples of *left* cord paralysis due to organismal toxins. In only one instance did a chemical factor (gout) cause the same lesion (Case VI).

5. If, therefore, one excludes Cases IV (aortic aneurysm), VII (pulmonary tuberculosis) and I, II, III, XIV in which the cord(s) was stabilized by a local mechanical lesion, there remain eleven instances of *left* cord paralysis and only one in which the right was affected.

While engaged in the preparation of this contribution, I met my friend Sir Frederick Hobday, F.R.C.V.S., and seized the occasion to ask him (*a*) the initial cause of " roaring " in horses and (*b*) which cord was the more frequently paralysed. With regard to the first query, he said that influenza and strangles were by far the most common sources of infection, and that in 4,300 operations for the cure of roaring, the *left* cord was paralysed in every case. He added, "If there was an exception in favour of the right cord, I cannot remember it." Furthermore, it was of interest to hear that, although, as in human beings, the left recurrent nerve of horses also passes under the aortic arch, they rarely suffer from aneurysm of that vessel. In addition to the blood-borne factors he mentioned, there was a small residue of his cases (roaring) in which the left cord was paralysed by becoming involved in a mass of enlarged glands in the neck.

So far, attention has been directed only to certain structural lesions and blood-conveyed poisons which may lead to vocal cord paresis, but it should be remembered that the same defect can be produced by what an Irishman might term negative factor ! This could readily be exemplified by the neuro-paralyses which follow a prolonged and insufficient supply in the daily foodstuffs, of certain essential vitamins.

Some Clinical Aspects of Vocal Cord Inaction

Perhaps the most flagrant example of avitaminosis is the disease known as beri-beri which is endemic in tropical climates of the Far East, where milled or polished rice is the staple article of diet amongst the poorer populations. Food thus prepared is deprived of the anti-neuritic constituent (B_1) or Vitamin B, and according to Sir Edward Mellanby the anti-infectious elements of Vitamin A. During the Great War, many amongst our troops in the Dardenelles became all too familiar with the symptoms of beri-beri which not only caused complete paralysis of the legs but the paralysis causes loss of voice (vide *Food, Health, Vitamins*, by Dr. R. H. A. and Mrs. Plimmer, Longmans, Green & Co.). Unfortunately, no mention was made of the laryngeal conditions observed (?) in such cases.

Although it is highly improbable that laryngologists practising in Great Britain will have opportunities for providing such information, the subject of avitaminosis has been introduced because it is quite conceivable that a persistently low average of any of those food essentials may suffice to activate some hitherto latent paralytic factor which might involve a vocal cord.

There now remain two main questions to be answered :—

1. What is the intimate neuropathology of vocal cord paralysis when caused by (*a*) a local lesion involving the extracranial course of a recurrent laryngeal nerve, and (*b*) blood-borne bacterial toxins or chemical poisons ?

In the first category—of which aortic aneurysm is a familiar example—pressure induces anæmia followed by degenerative atrophic changes in that portion of the nerve in immediate contact with the primary lesion. (For this explanation, I am indebted to a neurological expert.)

But, in the case of circulatory poisons, peripheral neuritis would seem to be the essential pathology of the paralysis. This, be it said is only my personal view, but the following brief observations appear to lend it considerable support.

Laryngologists, no less than physicians, are more or less familiar with its manifestations in post-diphtheritic palatal and/or vocal cord paralysis (*vide supra* Cases XV, XVI, XVII), while the same type of neuritis is responsible for the characteristic muscular palsies of lead, arsenic, alcohol, and uric acid (Case VIII) poisons.

One of the most striking features in several of the clinical

histories (*vide supra*) was the comparatively sudden onset and equally rapid disappearance of cord paresis. If that be of frequent occurrence in other regions, my own experience of peripheral neuritis would be a typical example. During a summer holiday, my sleep was disturbed by severe pain in the muscles of the right shoulder girdle and arm. It was so intense and disabling that I returned home the next day and placed myself under the care of a neurological colleague who did much to relieve the subjective discomforts. About five weeks later, and within forty-eight hours, all the symptoms disappeared completely.

2. Why is the left cord so much more frequently paralysed than the right ? Eleven to one in the preceding case records !

On the general principle that the vulnerability of a nerve is in direct proportion to its length, the longer intrathoracic course of the left recurrent laryngeal nerve obviously exposes it to the extra risks of aneurysmal dilatations of the aortic arch, peripheral extension of a mid-œsophageal cancer or its secondary deposits in some of the neighbouring mediastinal glands, and the more extensive pleural surface on which the nerve may become involved by a post-inflammatory adhesion.

But these are anatomical gauntlets and, as such, do not explain why the left nerve is more susceptible than the right to such blood conveyed factors which have been mentioned.

In these circumstances, does its mere length, *ipso facto* imply increased sensitivity, or are the left side structures congenitally less resistant than those on the right ? If not, why should so large a proportion of deafness caused by otosclerosis begin in the left ear ? Here, perhaps, one must await the answer of morphologists, or of those who have an equally expert knowledge of Comparative Anatomy.

Conclusion

These questions bring to its close a self-imposed task of which the main object has been to widen our view with regard to the pathology of those cases of vocal cord paralysis which are not caused by some local factor.

The time spent in its preparation will have been worth the while even if the clinical records, or comments on them, have only proved that the prognosis of such paralyses is by no means so unfavourable as physicians and the majority of

laryngologists have previously imagined. On the other hand, it would be too much to expect that peripheral neuritis as the neuro-paralytic factor will receive unlimited assent. In the meantime, unless and until a better explanation be forthcoming I can only say, as so often do the second judges in the Court of Appeal, " I have nothing further to add."

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In zwei frühern Arbeiten behandelte Verfasser die Frage der linksseitigen Stimmbandlähmung. Die weitern Erfahrungen der letzten zehn Jahre führten ihn dazu seine Ansicht zu ändern. Bei den vorliegenden Fällen stellte es sich heraus, dass die Heiserkeit oder auch weniger ausgeprägte Veränderungen der Stimme auf ein unbewegtes, im Uebrigen normales Stimmband zurückgingen, ohne dass eine erhebliche Veränderung oder Erkrankung am Ursprung, im Verlauf oder in der Verzweigung des entsprechenden Rekurrens vorhanden war. Nach jeder Krankengeschichte gibt Verf. seine eigene Zusammenfassung der wahrscheinlichen Ursache.

. . . .

L'auteur a exposé dans deux articles précédents la question de la paralysie de la corde vocale gauche. Au cours de ces dix dernières années, de nouvelles expériences ont modifié ses vues. Dans les observations actuelles, la raucité, ou quelque modification moins définie de la voix, était dûe à l'immobilité d'une corde vocale par ailleurs normale et ne pouvait pas être attribuée à une lésion relativement importante, intéressant l'origine, le trajet ou la distribution du nerf récurrent correspondant. Après chaque observation, l'auteur donne son propre résumé et l'explication probable.