The commonest tumour of the cerebrum is a glioma and its removal is impossible. The relief which may be obtained by decompression is, however, sometimes very considerable, and of this the following case furnishes an example.

A woman, aged 38, was sent to me with a history of severe frontal headaches extending over six years. A year before I saw her she suddenly fell down unconscious and had a series of violent convulsive attacks starting in the left arm. From this she recovered completely. It had for some time been noticed that she was somewhat unstable mentally, acting with excitable levity. She occasionally had a tremor in the right arm and leg, and vision was failing.

She was an excitable woman in abnormally high spirits. She had severe papilloedema, most marked on the right, but no changes in motion, sensation, or reflexes were discovered. A deep glioma of the right frontal region was diagnosed, and a large decompression in this region was performed, the brain being under very high pressure. She recovered completely, her headaches disappeared, and she regained her normal health and vision; even the fundi became normal. For eighteen months she remained in excellent health, though a left hemiparesis and an enormous hernia very gradually developed. She then quite rapidly became comatose and died.

Although a glioma cannot be removed it may degenerate in the centre and become cystic, and in such a case it is sometimes possible by aspiration of the cyst to give considerable relief to the patient. If the cyst is at all superficial and can be treated with formalin the relief may extend over a considerable period; in some of these cases the introduction of radium has given good results. In such cases a decompression not only relieves the pressure but enables the cyst to be aspirated with facility. The following is an example:

A man, aged 44, was referred to me by Dr. Riddoch with the story that six months previously he had had a sudden attack of giddiness, followed by pains in the back of the neck and difficulty in speaking, which lasted for a few minutes. Since then he had had a few similar attacks, and six weeks before I saw him, after one of these, the right arm began to shake and the right side of the face to twitch. When this had passed off the arm remained weak and the difficulty in speech persisted. He was an intelligent man with marked apraxia and aphasia. There was slight weakness of the right side of the face and tongue, and spastic paresis of the right arm and leg with increase in the deep reflexes. He rapidly became worse, complete right hemiplegia developed, he was unable to move his tongue, and had increasing difficulty in swallowing. He became very drowsy and the respirations stertorous. Left temporal decompression was performed and a large cyst discovered lying deeply in the left frontal lobe, from which 44 c.cm. of turbid fluid was withdrawn. He made a remarkable recovery. His speech commenced to return in four days and he regained full power in his limbs.

The only removable tumours which are at all common in the region of the cerebral hemispheres are the endo-theliomata. These arise from the inner surface of the dura, probably from the lacunae of the anterior longitudinal sinus. Sometimes they are firm, rounded, fibrous tumours, often, however, with central degeneration; sometimes they are soft and diffuent. They may have a pedicle or they may be sessile on the inner surface of the dura. The bone over them is very vascular, and they have a curious power of stimulating its growth, so that external bony tumours may appear on the skull. They may, indeed, invade the bone, with which they thus become indissolubly connected, but in no circumstances do they invade the brain; they press into its substance, and their removal may at first leave a large cavity, but in a few moments the brain returns to its normal position and the cavity is entirely obliterated.

Theoretically these tumours should be completely removable and should never recur, but it is not always possible to obtain this result. In the soft type the tumour may break up so far that there can be no certainty of its complete removal, and in the sessile variety its connexions with the superior longitudinal sinus may be so complete that only a resection of the sinus, with the grave risks which that involves, could ensure total ablation. One case of the latter condition has already been mentioned, and of the former the following is an example:

In October, 1921, I operated upon a woman, aged 25, who for eighteen months had suffered from morning headaches of increasing severity, and latterly accompanied by vomiting. She had had attacks of tingling and numbness in the fingers of the left hand, spreading to the shoulders and to the left side of the tongue, and loss of sense of position in the fingers of the left hand.

The right Rolandic area was exposed by an osteoplastic flap, and a large, soft, purplish tumour was found arising from the inner surface of the dura over the middle of the right Rolandic area.

On opening the dura a mass of diffuent material about the size of a tennis ball oozed out, the cavity in the brain being almost instantly obliterated. The dura was removed as widely as was practicable and the osteoplastic flap was closed. She made an uninterrupted and complete recovery.

In April, 1924, after three and a half years of perfect health, she suddenly had a succession of violent fits, starting with tingling in the left fingers, but becoming general, and accompanied by loss of consciousness. The papilloedema, which had entirely disappeared, was found to have returned. Exploration showed masses of soft growth protruding through the old trephine holes in the bone, which was itself widely invaded by soft vascular growth. The bone was widely resected and the growth removed as completely as was possible, although it extended up to the middle line. She again recovered completely, and so far has remained in perfect health. It is possible that the use of x rays over the large opening left in the skull may enable us to prevent further recurrence.

It will thus be seen that although only in a very limited number of cases can we expect to effect a complete cure by surgical interference, there are many in which we can give considerable relief. The most important factors in success are early diagnosis and precise localization; both should be effected before general symptoms have developed and before an increased intracranial pressure has added to the difficulties and dangers of an operation. There is no field of surgery in which so much depends upon the close co-operation of the physician and the surgeon, upon the enterprise of the former and the restraint of the latter.

An Address

ON

MASSIVE PULMONARY COLLAPSE.*

BY

A. BERTRAM SOLTAU, C.M.G., C.B.E., M.D.,
M.R.C.P., F.R.C.S., K.H.P.,

PHYSICIAN, SOUTH DEVON AND EAST CORNWALL HOSPITAL, PLYMOUTH.

It might be supposed that the subject of pulmonary collapse is largely of academic interest, but the clinical manifestations are of great importance, both to the physician and to the surgeon, and a prompt recognition of the physical signs and the symptoms, and where possible their prevention or their rectification, is therefore of great value.

PHYSIOLOGICAL INTRODUCTION.

Before proceeding to discuss the causation of pulmonary collapse a brief recapitulation of the essential anatomical and physiological facts of respiration may be helpful. It is customary to regard the lung as a single organ, expanding as a whole, and to disregard a possible significance of its division into lobes. Keith¹ has, however, shown that the great fissure is of fundamental importance, in that it separates the lung into areas, the method of expansion of which is distinct, the upper lobe being expanded by the mechanism of the upper ribs, and the lower lobe by a compound mechanism formed by the diaphragm and lower ribs. Further, the extensibility of the lung is not uniform in degree, but varies with the three anatomical zones into which it is divided. These are (a) *the root*, practically non-extensible; (b) *the intermediate zone*, containing vascular and bronchial ramifications with some extensible tissue between; and (c) *the outer zone*, consisting entirely of expansile tissue. Expansion takes place by the moving apart of the rays of the inextensible structures, and the filling out of the intermediate and outer expansile areas, a mechanism which Keith has likened to the opening of a Japanese fan.

It is impossible here to enter into any detail of the muscular mechanism brought into play in respiration, and a somewhat dogmatic outline must suffice.

The upper part of the thoracic cavity is conical in shape, the lid being formed by the first ribs and the manubrium sterni. This lid is held immobile by the scaleni, and acts as the fixed point from which the upward movement of the ribs originates. In the main the expansion of the upper lobe is brought about by the lifting of the five upper ribs, with an increase in the antero-posterior thoracic diameter, owing to the axis of rotation of these ribs being set almost at right angles to a vertical section. Each successive rib

* Delivered before the Plymouth Medical Society.

moves through a rather wider space than the one above; hence the undulating expansion described by Hoover.² The lifting of the ribs is the result of contraction of the external intercostals. The contraction of the diaphragm has a very slight expansile effect on the upper lobe, for the crura, being attached to the root of the lung through the pericardium, will on contraction displace the pulmonary hilus downwards, and so help the ventilation of the apex.

The lower part of the thorax from the fifth rib downwards is cylindrical in form, and for its expansion depends in part on the intercostals and in part on the diaphragm. The external intercostals carry on the same lifting process as already described, but with the alteration of the axis of rotation of the ribs, the angle with the vertical mid-plane becoming progressively more acute, the resultant movement is rather in a lateral than in an antero-posterior direction.

The contraction of the diaphragm is the other great factor in the ventilation of the lower lobes. Attached behind to the lumbar vertebrae, laterally to the lower edges of the six lower ribs, and in front to the ensiform cartilage, the fibres converge to their insertion in the central tendon. At rest two domes are formed below the bases of the lungs, the outer wall of each being in contact with the parietal walls. Contraction shortens the fibres, flattens the domes, and by so increasing the vertical diameter of the thorax permits the lungs to descend and expand. The diaphragm and the intercostals are in opposition in their pull on the lower ribs, for the former tends to close the subcostal angle and the latter to open it. The flatter the dome of the diaphragm the more direct is the pull it can exert, and the more force it will bring into play on the costal margin (Hoover²). This point is of considerable clinical importance. Depression of a diaphragmatic dome, either by pleural effusion or cardiac enlargement, will increase its mechanical efficacy, and this is shown by a reduction in the subcostal angle, instead of its increase as in normal respiration, in which the outward pull of the intercostals can overcome the inward pull of the diaphragm (Macleod³). A very critical study by Sir Charlton Briscoe,⁴ in his Arris and Gale Lectures, of the action of the diaphragm will repay close study by those interested in this subject. He differentiates the contraction of the diaphragm into two parts—the crural, dependent solely on the phrenic nerves for stimuli, and the costal, which obtains part of its nerve supply from the intercostal nerves. He has further classified patients into three types, according to their manner of breathing. The A type depends almost entirely on the crural contractions for diaphragmatic respiration. In the C type, at the other extreme, the costal contractions are the more prominent. The adoption of the supine position will materially affect diaphragmatic respiration, and this has, as will be seen later, a profound influence on the production of collapse.

THE CAUSATION OF PULMONARY COLLAPSE.

It is to William Pasteur that the credit is due for first noting and describing the phenomena which he described as "post-operative massive collapse of the lung," in 1908.⁵ Several other communications by him followed, and he gave a general review of the subject in 1911.⁶ It was not, however, until the war led to a concentrated study of chest wounds that collapse came to be recognized as a frequent occurrence, for explanation had to be found for the many cases in which signs were present which could not be attributed to any of the generally accepted pathological conditions. In all the published writings on gunshot wounds of the chest references will be found to pulmonary collapse (Herringham,⁷ Soltau and Alexander,⁸ Gask and Wilkinson,⁹ J. R. Bradford,¹⁰ Soltau,¹¹ Bradford¹²).

It is obvious that the collapse of a lung, in whole or in part, may be brought about by various causes. A complete obstruction of the main bronchus, as occasionally occurs from inhaling a foreign body, must inevitably lead to such a condition, and this fact led to the theory that collapse was caused by the accumulation of bronchial secretion to such an extent as to plug the bronchi (Elliot and Dingley¹⁷). Such a condition, however, has not been commonly found in *post-mortem* examinations. Moreover, in many cases a lung has been profoundly collapsed during

life without the patient having any expectoration or other sign of increased bronchial secretion. Cases of purulent bronchitis with great excess of secretion do not appear ever to develop a massive collapse, though small collapsed areas may be seen *post mortem*. The collection of secretion cannot therefore be accepted as a complete explanation, though in some cases it may play a small contributory part.

Pasteur himself regarded the condition as being due to diaphragmatic paralysis, and there is increasing evidence that inhibition of the activity of that muscle forms an essential part of the process of collapse. X-ray examination of these cases invariably shows that the diaphragm on the affected side is raised, often to a higher level than that reached in the extreme expiratory position. It has even been seen to move in the reverse direction, rising with inspiration owing to the increase of intra-abdominal pressure from the descent of the unaffected lobe. Moreover, frequent opportunity was afforded during the war of observing the diaphragm through a hole in the chest wall—from wounds or on the operating table—and the flaccid, atonic condition of the muscle was obvious.

It is not so easy to determine why the diaphragm is paralysed, and this question is still under debate. The present writer, with Dr. Alexander,⁸ advanced the view that it was a reflex paralysis, the afferent impulses being conveyed to the respiratory centre from the focus of irritation by means of the vagus, and the efferent by the phrenics. They pointed out that collapse was not of necessity associated with injury to the lung or to the thoracic wall, but might occur, as Pasteur had originally noted, after abdominal injuries quite remote from the diaphragmatic region. A penetrating wound of the pelvis, for instance, exhibited a very typical picture of pulmonary collapse. In military experience certainly by far the greater number of cases were directly associated with injuries, not necessarily penetrating, of the chest wall or lung. In civil experience the majority of cases are a direct sequel to abdominal operations, particularly those on the upper abdomen. In the writer's experience operations involving the liver and bile passages are especially likely to be followed by collapse of the lung.

Briscoe⁴ advances an alternative view as to the origin of the paralysis, regarding it as merely an exaggeration of the respiratory changes which take place when the supine position is adopted. In this position the costal type of diaphragmatic respiration is largely suspended, particularly in those patients of type A whose respiration is mainly crural in origin. Subsequent irritation of the crura or of their pleural covering will lead to inhibition of the crural respiration, and hence to paralysis and complete deflation. Further observation is needed to determine the relative importance of these two views. Whatever the actual mechanism which inhibits the diaphragm, the result is a cessation of ventilation of the lung, with speedy absorption of the residual alveolar air, and so complete collapse. Falconer¹³ supports Briscoe's view and records the comparatively frequent occurrence of massive collapse in prolonged malarial cases. He was inclined to regard the condition as being caused by perisplenitis. Watson and Meighan¹⁵ have recorded cases of diaphragmatic paralysis which they attributed to a functional disturbance of the phrenic centre. It would be interesting to know whether any observations have been made in the cases, recently described, of very rapid breathing as a sequel to encephalitis lethargica, to note the condition of the diaphragm.

Whilst the study of pulmonary collapse from injury was being studied a further very interesting fact came to light—namely, that the uninjured lung may frequently share in the condition, a phenomenon to which the term "contralateral collapse" was applied. This condition has been seen both with penetrating and non-penetrating wounds of the chest. The radiograms exhibited, taken by Captain Donaldson, show an extreme degree of massive collapse of the right lung, resulting from a severe blow to the left thoracic parietal wall. The writer recently saw a similar case in which the shaft of a cart had inflicted a heavy blow on the left side, and the right base was collapsed. Curl¹⁴ has described in some detail the contra-

lateral signs associated with chest wounds. These cases of contralateral collapse are difficult to explain without assuming a nervous reflex.

Finally, the well known experience of being "winded" by a sudden blow may be due to temporary paralysis of the diaphragm. Here the immediate onset must preclude any inflammatory cause. The happy conjunction of an x-ray plant and a "winded" patient may settle the point.

VARIETIES OF COLLAPSE.

The most common condition to find is massive collapse involving the whole of the lower lobe. More rarely the whole of the lung may be involved, and very occasionally the upper lobe alone is affected. In cases where a part only of the lower lobe is collapsed a wedge-shaped area with the base downwards will be found, giving the characteristic signs. Collapse may be homolateral, or, in cases of injury, contralateral. In civil practice collapse following surgical operations most commonly affects one base only, but it may be bilateral, each side becoming involved successively or simultaneously.

PHYSICAL SIGNS.

These are not unequivocal, and some practice is required to make certain of the diagnosis, for in many respects there is a close resemblance to the physical signs of pneumonia. Observation is of the greatest help, for the *retracted* immobile chest wall affords a striking contrast to that of pneumonia, in which with immobility the appearance of fullness is so frequently noticeable. The difference in the ribs in the two cases may be compared to the appearance of a concertina when closed and when extended.

Palpation is the critical diagnostic test, for by that means the position of the apex beat may be determined. The most definite and constant sign of collapse is displacement of the apex, and this always takes place towards the collapsed area. The displacement may be extreme, the apex being found well over towards the axilla, and frequently lying at a higher level than the normal.

This alteration may take place with great rapidity, the apex having been found to have moved two inches in the course of a few hours. On percussing the chest a marked alteration in the note will be elicited. With complete collapse there is absolute dullness, so definite as to suggest the presence of fluid. The position of the apex beat, however, at once eliminates the possibility of effusion. On the left side the increase upwards of the gastric resonance will indicate the high level of the diaphragm, and so suggest paralysis of that muscle. The auscultatory sounds may vary very greatly, being modified both by the degree of collapse present and with the phases through which the condition passes.

In a typical case followed from its commencement, at first breath sounds are practically absent, or only the faintest respiratory "whisper" will be heard. This absence of breath sounds is accompanied by absence of vocal resonance. We are listening to a "dead" lung through which no bronchial air is passing. The sign-complex is therefore a retracted immobile chest with raised diaphragm, dull on percussion, and exhibiting neither breath sounds nor vocal resonance, and with the heart displaced towards the dullness. In the second stage there is a dramatic alteration. The percussion note is still dull, but the breath sounds are tubular or amphoric, and vocal resonance is greatly increased. Ventilation is commencing and the bronchial respiration becomes audible.

The clinical picture resembles a pneumonic consolidation, except for the shape of the chest and the position of the apex. In the final stage dullness becomes less pronounced, the tubular breath sounds are less definite, and frequently moist adventitious sounds are present. Air is commencing to enter the alveoli and a certain excess of secretion may be present. With the expansion of the lung the heart returns gradually to its normal position. This phase is frequently confused with bronchitis, and probably not a few of the cases of so-called post-anaesthetic bronchitis are really in the final stage of massive collapse. Pyrexia is not uncommon, but does not reach a high degree. It is to be

expected that a bronchial infection will frequently complicate collapse, and even a true pneumonic consolidation may occur in the collapsed lung.

SYMPTOMS.

Massive collapse with unmistakable physical signs may produce very few symptoms. A certain degree of dyspnoea, more marked on exertion, is usually present, and is, of course, much more pronounced if both lungs are involved. Cyanosis is not a marked feature, for the unaffected lung area is ample to secure sufficient oxygenation whilst the patient is at rest. The writer has, however, not infrequently noticed that the colour of the face is a brick red, and has on more than one occasion ventured a diagnosis of collapse based on this appearance, and has found it confirmed on further examination. The absence of cyanosis may be explained by Haldane's¹⁶ reasoning, that where a lung, by consolidation or collapse, has ceased to have any pulmonary circulation, only the bronchial persisting, the whole of the venous blood is oxygenated in the unaffected areas. Where, however, the pulmonary circulation is partially open, venous blood will circulate through lung area which is not capable of oxygenating it, and therefore a mixed stream of oxygenated and non-oxygenated blood will reach the systemic circulation, causing cyanosis.

Cough of a weak, irritating type is frequently present. It is not usually productive—in part owing to lack of pressure from behind, as the diaphragm is out of action, and in part also because secretion is not necessarily increased.

DIAGNOSIS.

The diagnostic signs have been detailed already, and it is only necessary to refer very briefly to the main points. The appearance of the chest and the position of the apex beat are the two cardinal signs to which attention should be directed. If these be remembered, confusion with either pleural effusion, pneumonia, or bronchitis will not arise. In military practice the presence of a coexisting haemothorax introduced disturbing signs, which will practically never arise in civil practice. It should not be forgotten, however, that a pneumonic consolidation is not unknown in a collapsed lung, more particularly as reinflation is commencing. So, also, an infective bronchitis may be associated with collapse. It is hardly necessary to refer to the great assistance in diagnosis which x-ray examination gives, and in cases of doubt this assistance should, where possible, be sought.

TREATMENT.

It is obvious that very little can be done in the way of active treatment. In cases in which dyspnoea is a symptom oxygen is essential. This should be given practically continuously in measured quantities, by means of a Haldane oxygen apparatus. In cases requiring prolonged or exhausting dressings oxygen should be given as a routine during the dressings. The importance of Sir Charlton Briscoe's investigation lies in the fact that the supine position favours collapse. To the surgeon the value of placing his patient as soon as possible in the semi-upright position will appeal. It is hardly too much to say that all operations on the upper abdomen should be nursed in this position, as soon as post-operative shock has passed off. Nursing homes still are frequently ill provided with comfortable and easy means of raising patients into this position and maintaining them there. The Fowler frame, introduced during the war into casualty clearing stations, was an admirable and simple mechanical device for attaining this end.

The object of this communication is to call attention to a condition which occurs far more frequently than is supposed, and which has not received much attention in the general medical press.

SUMMARY.

1. Massive pulmonary collapse is a definite clinical condition.
2. It is caused by inhibition of the diaphragmatic action, whether from reflex or inflammatory causes.
3. Its onset is favoured by the supine position.

4. It follows direct trauma of the chest wall or lung, and surgical operations on the abdomen, even remote from the diaphragm.

5. It gives definite physical signs, which may simulate pneumonia or pleural effusion.

6. The differential diagnosis depends on determining the position of the apex beat and the diaphragm.

7. It may, in cases of injury, affect the lung on the side remote from the trauma.

8. Its occurrence, whilst of itself not necessarily of grave import, yet places another burden on a patient already struggling with injury or disease.

9. Treatment should be preventive.

REFERENCES.

- ¹ Keith: *Further Advances in Physiology*, 1909. ² Hoover: *Arch. Int. Med.*, 1917, xx, 701. ³ Macleod: *Physiology and Biochemistry*, fourth edition, Chapter XXXVII. ⁴ Briscoe: *Quart. Journ. Med.*, 1919-20, xiii, p. 293. ⁵ W. Pasteur: Bradshaw Lecture, *Lancet*, 1908, ii, 1351. ⁶ W. Pasteur: Oration before Medical Society of London, *Lancet*, 1911, i, 1329. ⁷ Herringham: *Quart. Journ. Med.*, 1916-17, x, 79. ⁸ Soltan and Alexander: *Ibid.*, 259. ⁹ Gask and Wilkinson: *BRITISH MEDICAL JOURNAL*, 1917, ii, 10. ¹⁰ J. R. Bradford: *Quart. Journ. Med.*, 1918, xii, p. 127. ¹¹ Soltan: *War Medicine*, American Red Cross Society, 1918, vol. ii. ¹² J. R. Bradford: *Ibid.* ¹³ Falconer: *Quart. Journ. Med.*, 1919-20, xiii, 25. ¹⁴ Curl: *Ibid.*, 1918, xii, p. 161. ¹⁵ Watson and Meighan: *BRITISH MEDICAL JOURNAL*, 1919, i, 303. ¹⁶ Haldane: *Organism and Environment*, 1917. ¹⁷ Elliot and Dingley: *Lancet*, 1914, i, 1305.

ON THE POSSIBILITY OF RESTORING MOVEMENT TO A PARALYSED VOCAL CORD BY NERVE ANASTOMOSIS.*

(An Experimental Inquiry.)

BY

LIONEL COLLEDGE, M.B., F.R.C.S.,

SURGEON, EAR AND THROAT DEPARTMENT, ST. GEORGE'S HOSPITAL.

THE following series of experiments were carried out by Sir Charles Ballance with the collaboration of the writer. The experiments were performed at the National Institute for Medical Research, Hampstead, and were made possible by the facilities granted by the Medical Research Council. Although final conclusions have not been reached, certain definite results have been obtained, and it is at the suggestion of Sir Charles Ballance that this description of the research has been written.

Experiments on the effects of an anastomosis of the recurrent laryngeal with another nerve by previous observers do not appear to have given very definite results, probably owing to the difficulty of accurate observations on the movements of the vocal cords in animals without killing them at the end of the experiment. This is now easily overcome by using a direct vision laryngoscope. The larynx of a monkey and the movements of the vocal cords can be not only seen but carefully studied through the spatula of Chevalier Jackson, employing the model designed for the child. Direct observation has therefore taken the place of inferences drawn from the presence or absence of such symptoms as stridor, hoarseness, or roaring in animals. It has even been possible, although certain technical difficulties had first to be overcome, to record the movements of the vocal cords on a cinematograph film.

The results of the experiments may be divided into three groups.

I.—ANASTOMOSIS WITH THE VAGUS.

The first nerve employed was the vagus. Chevalier Jackson, in his book on endoscopy, suggests that an anastomosis between the vagus and the recurrent laryngeal might cure paralysis of a vocal cord. Since the recurrent laryngeal nerve passes down the neck in the trunk of the vagus, such an anastomosis would be most likely to succeed if the cut end of the recurrent laryngeal were united to that part of the vagus which contains the fibres of the recurrent laryngeal, before they have left the main trunk. There is, however, no means of distinguishing the fibres of the recurrent laryngeal from the rest of those which form the trunk of the vagus, so that there would be only a small chance of effecting such a reunion.

* An illustrated article by Sir Charles Ballance on experiments in which the facial and recurrent laryngeal nerves were anastomosed with other nerves was published in the *JOURNAL* (1924, vol. ii, p. 349).

Actually the end of the recurrent laryngeal was implanted into the side of the vagus in two goats, a rhesus monkey, and a baboon; but the last animal died of pneumonia three weeks later, which suggests that the operation may not be devoid of risk. There were therefore three effective experiments in this group.

In a large male goat the vagus was nearly half divided and the cut recurrent laryngeal sutured to the proximal lip of the incision. At the end of ten months no movement of the vocal cord had been observed, though it appeared to have become straight and tense. This effect, however, may have been produced by contracture of the intrinsic muscles of the larynx rather than by the return of the muscular tone. On exposing the anastomosis the recurrent laryngeal was found kinked and involved in scar tissue, nor was any movement of the cord produced by stimulation of the trunk of the vagus, the anastomosis, or the recurrent laryngeal itself. The experiment repeated on a young goat gave negative results at the end of six months.

In the rhesus monkey the same operation was done and the corresponding vocal cord was seen to be paralysed. At the end of four months no movement had returned though the cord was more tense. The anastomosis was exposed at the end of seven months, and abduction of the paralysed cord was obtained by stimulation (a) of the trunk of the vagus, (b) the site of the anastomosis, and (c) the recurrent laryngeal nerve.

The excursions of the cord were not quite so wide as on the sound side.

It appeared, therefore, that the muscles were innervated from the vagus, although no spontaneous movement occurred, and Professor Starling suggested that if the animal were partially asphyxiated the forced inspiration so produced might be so powerful that the muscles would receive a strong enough nervous impulse to cause a contraction. This experiment was tried at the end of nine months, and the cord was seen to abduct several times, though not so widely as on the opposite side. The same effect was seen at the end of twelve months, but was less after fifteen months, and has disappeared after seventeen months, and still has not reappeared.

It may be concluded from these experiments that, although a nervous connexion may be re-established by a recurrent laryngeal vagus anastomosis, it is useless to expect return of spontaneous movement in the corresponding vocal cord.

II.—ANASTOMOSIS WITH THE DESCENDENS NONI.

In another series of experiments the recurrent laryngeal nerve was anastomosed end-to-end with the descendens noni. This operation was done on four rhesus monkeys.

In the first experiment the recurrent laryngeal was anastomosed end-to-end with the descendens noni and the distal end of the descendens noni implanted into the side of the hypoglossal. Before the suture was completed it was observed that stimulation of the distal end of the recurrent laryngeal caused abduction of the cord. This animal died from bites received from another monkey six weeks after the operation, so that no result was obtained.

In the second experiment the same operation was done on the right side, except that the distal end of the descendens noni being very small was left free. At the end of two months there appeared to be increased tension in the cord but no spontaneous movement. Stimulation of (a) the hypoglossal proximal to the origin of the descendens noni, (b) the descendens noni, (c) the anastomosis, all caused abduction of the cord, showing that a nervous impulse could pass through the anastomosis. At the end of four months there was still no spontaneous movement in the cord, but on partial asphyxiation abduction of both cords followed but soon ceased. At the end of five months, and again at the end of seven months, there was still no movement of the cord on tranquil respiration, but wide excursions of both cords followed partial asphyxiation and lasted for two minutes. The movements of the right cord gradually ceased as the effects of the asphyxiation passed away. The animal became ill at the end of nine months and was killed, but it was noted that still there were no spontaneous movements of the right cord.

In the third experiment the animal had to be killed at the end of seven months; the results were exactly the same as in the second experiment.

In the fourth experiment the descendens noni was anastomosed end-to-end with the recurrent laryngeal. The distal end of the descendens was implanted into the side of the phrenic. No spontaneous movement returned to the vocal cord, but when the animal was killed at the end of three months stimulation of (a) the hypoglossal proximal to origin of the descendens noni, (b) the descendens noni, and (c) the anastomosis, all caused abduction of the vocal cord.

The results of these experiments with the descendens noni show that, as in an anastomosis with the vagus, the muscles controlling the movements of the vocal cord become innervated, but no movement occurs in the cord because the appropriate impulse does not pass along this route.