head of the humerus was not well covered by the muscle in front. The triceps and biceps muscles were greatly atrophied, especially on the right side, as is seen by the measurements around the middle of the upper arm, which were on the right side $7\frac{1}{2}$ in., and on the left $8\frac{1}{2}$ in. The right supinator longus was very weak and wasted, but the left was only slightly affected. In the muscles on the radial side of the left forearm there was a small hypertrophied portion, about $\frac{3}{4}$ in. in length, which felt softer than the muscles around when they were contracted. The grasp of the right hand was not so strong as that of the left, but otherwise the muscles of the hands appeared normal. The pectoral muscles were greatly affected in rather a peculiar manner. The clavicular portion of the pectoralis major, on the right side, did not appear to be much affected, but the sternal portion was greatly wasted and could not be contracted voluntarily. On the left side the condition was reversed, the clavicular portion being wasted and the sternal portion contracting well and standing out prominently. The sterno-mastoid muscles were also greatly atrophied, and equally so on the two sides, so that there was a distinct hollow above the sternal end of each clavicle. The supraspinati muscles were unaffected. The infraspinatus muscle on each side, but especially on the left side, was so greatly hypertrophied as to cause quite a prominence. The scapulae were "winged," which became very marked when the arms were raised in front. The serratus magnus, rhomboids, and the levator anguli scapulae on each side were distinctly weak. The back and the sides of the neck looked to be very swollen, which appearance was produced by the great hypertrophy of the upper portion of the trapezius muscles. These muscles were greatly thickened on each side of the neck, and their anterior borders were very prominent, while on the left side of the neck there was a small tumour, about the size of a hen's egg, which was buried in the muscle and which had the consistence of a lipoma. The other muscles of the back appeared to be normal. In the legs the only affected muscle was the left tibialis anticus which was atrophied. There was no weakness of the muscles of the calves and the patient could easily and for a considerable length of time stand on tip-toe. The movements of the muscles of the face were not good and the face itself seemed to be wanting in expression. The patient could not screw up his eyes tightly, pointing to some weakness of the orbicularis palpebrarum muscles, which are so usually found affected in this disease when commencing earlier in life. He also could not whistle, although previously he had been able to do so. The contractions of the masseter muscles were distinctly weak. The tongue was protruded in the middle line and there was no tremor. There was no affection of the muscles of deglutition and no alteration in the voice. The gait was normal, except that the patient bore his weight more on his right than on his left leg. When placed flat on his back, the patient was unable to get up without rolling over. He then got on his hands and knees and regained the upright position in a normal manner.

Electrical Reactions.—These in the affected muscles were as follows. There was diminished excitability to the faradic and galvanic currents, and the response to the cathode closing of the circuit was readier than that to the anode closing. The reaction of degeneration was, therefore, not present.

Sensation.—Tactile, painful and thermal sensations were normal. Muscular sense was also good, as the patient knew the position of his limbs, and could estimate accurately the differences in the weights of small objects of the same volume.

Reflexes.—The knee-jerks were present, but hard to obtain. There was no movement of the toes on stimulating the soles of the feet; the biceps and wrist-jerks were present, and the abdominal reflexes were very sluggish.

Eyes.—The pupils were equal, and reacted to light and to accommodation. There was no strabismus or nystagmus, the discs were normal, and there was no diminution in the fields of vision.

Other Systems.—The pulse was regular, and of good volume; the arteries were not abnormally thickened. Urine, specific gravity 1032, acid, no albumen or sugar. Nothing abnormal was detected elsewhere.

Treatment.—The affected muscles were massaged once a day for about half an hour, and the patient was encouraged to use his arms.

Progress.—By August 9th there was a slight gain in strength in the arms and he was able to brush his hair by supporting his elbows on a table and by bending his head forward. He could also get his hand on to his head by swinging the arm up in front, but could only keep it up in this position by resting the hand against the back of his head. He could only abduct the arm so as to form about half a right angle with the side of his body. The painful sensations, which he had in the right arm, quite disappeared, but towards the end of August he suffered from neuralgia of the face, which was more severe on the right side. This very slight improvement in the arms continued for about a month, at the end of which time he was able to perform his toilet by various contrivances such as supporting one arm on the other or by laying his head on the table to brush his hair. The grasp of the right hand also seemed to gain a little in strength. From this time onwards the condition remained stationary, and he was discharged from the hospital on September 22nd, 1901.

The peculiar part about this case, and that which warranted its publication, was the hypertrophy, which was very obvious, of some of the muscles of the shoulder girdle, commencing at the age of 43. That this enlargement was a pseudo-hypertrophy cannot be absolutely proved without a microscopic examination of the muscle fibres. The diseased muscles were those that were chiefly used in the patient's occupation as a coal-porter. I cannot find an account of a single case in which this pseudo-hypertrophy began so late in life, although there are many recorded instances of myopathy with atrophy. On account of the distribution and grouping of the affected muscles, and also on account of the absence of the reaction of degeneration and of any form of altered sensation, there can be very little doubt that the muscles, and not any part of the nervous system, were the seat of the disease in this case.

The gain in strength which accompanied the massage was, without doubt, due to the increased development of the unaffected muscular fibres, together with an arrest in the progress of the disease, and not to any regeneration in the affected portions of the muscles.

CASE OF MOTOR APHASIA, WITH HEMI-ANÆSTHESIA, BUT NO HEMIPLEGIA.

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THE following case of motor aphasia presents four prominent features: Motor aphasia, right-sided hemianaesthesia, no hemiplegia, and paralysis of the tongue on the right side.

History.—An unmarried woman, aged 50, was admitted into the infirmary on November 22nd, 1899, being unable to speak. According to her friends she was taken ill fourteen days before admission, being seized in the street with what they called a "fainting fit." She did not, however, fall or lose consciousness, but walked home, a distance of over a mile. She was unable to say what had happened to her, and since that day had not been able to speak. She had always been a healthy woman, never having had a day's illness. There was no history of fits.

State on Admission.—She was in a condition of general weakness, but walked into the ward with only slight assistance, and did not show any inclination to fall to either side. She understood perfectly what was said to her, obeying at once all orders, protruding her tongue, raising her arm, or closing her eyes when told to do so. She could not speak except to say "Ah," although she made great efforts to do more. She had never been able to read or write.

There was complete loss of sensation all down the right side from the scalp to the foot (the conjunctiva, palate, and tongue included), ending abruptly at the middle line. The special senses of taste, smell, hearing, and sight were normal. The tongue when protruded pointed markedly to the right side. On the right side, the arm, leg, and chest were a little weaker than on the left side, the difference being, however, very slight, and only apparent when looked for. There was no dragging of the leg. The right patellar reflex was a little exaggerated, and the right pupil a little smaller than the left. Both pupils reacted to light and accommodation. The fields of vision were normal, there being no restriction.

The heart was normal in all respects, the pulse 72, tense, and regular the wall of the artery being thickened and degenerated. Urine, specific gravity 1020, acid; a faint cloud of albumen was obtained on boiling, which disappeared a few weeks later.

Progress.—The patient was kept in bed for a few days, much against her wish, but ten days after admission was walking about quite well except for her inability to speak. On January 20th, 1900, she could feel very slightly the prick of a pin on the right side of her scalp, face, and tongue; she was able to say "doctor" and to count up to five, the other women in the ward having encouraged her to practise. A few days later she could say a few more words.

On March 15th the sensibility of the head, neck, and hand on the right side was much greater, though still far from normal. The woman shortly afterwards left the infirmary, and I have not been able to learn anything about her.

The diagnosis is interesting, but not very obvious. In the first place, was the disease functional or organic? In favour of its being functional was the hemianaesthesia, ending abruptly at the middle line, which form of anaesthesia existing without hemiplegia is one of the most typical signs of hysteria. Against its being functional were: The absence of any restriction of the fields of vision which is a very constant accompaniment of neurotic trouble, the paralysis of the tongue, the presence of genuine motor aphasia and not aphonia, or loss of voice, the previous history, age, and appearance of the woman, together with the returning sensation of the face.

If therefore the lesion was organic, of what pathological nature was that lesion?

Since marked anaesthesia is much more common in softening than in haemorrhage,¹ it would seem more likely to have been a softening than a haemorrhage, but whether that softening followed embolism or thrombosis it is impossible to say. The sudden onset points to embolism; the absence of any heart trouble and the presence of the degenerated arteries point to thrombosis.

LOCALIZATION OF THE LESION.

Considering the two most prominent symptoms separately, we notice that hemianaesthesia may be caused by a lesion in any one of the following situations:

(a) The posterior third of the hind limb of the internal capsule, where the afferent fibres of general sensation ascend to the brain. Such a lesion without any hemiplegia is extremely rare, and when present is usually accompanied by at least partial paralysis of the leg, also by hemianopsia, disturb-

ance of hearing, and sometimes of taste and smell, none of which were present.

(b) The cortex. This would of necessity be an extensive lesion, and would involve the motor areas, causing considerable paralysis and probably twitchings.

(c) The subcortical area. That is somewhere in the white matter which lies between the cortex and the internal capsule (corona radiata), and this I consider the most likely position. C. L. Dana in his study of sensory localization found that total anaesthesia was either of functional or subcortical origin.¹

Applying the same tests to the other prominent symptom, the motor aphasia, I come to the same conclusion; for if the lesion had been in (a) the internal capsule, it must have been at the anterior end of the hind limb, at the genu, since it is here that the fibres to the tongue and those concerned in motor aphasia pass through. (b) The cortical centre concerned in motor aphasia is at the posterior end of the third left frontal convolution, but this is unlikely from the absence of paralysis or twitchings.

We are thus again compelled to fall back upon the subcortical area as the most likely position for the lesion; and this would also account for the very slight weakness down the right side. I do not attempt to explain the inequality of the pupils, which often occurs in perfectly healthy individuals.

I am, therefore, of opinion that the lesion in this case was a softening in the subcortical area of the brain.

This case was shown at the Liverpool Institution, in March 1900.

REFERENCE.

¹ Osler, *Principles and Practice of Medicine*, 4th ed., p. 1025.

The Croonian Lectures

ON THE

NATURAL HISTORY AND PATHOLOGY OF PNEUMONIA.

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LECTURE II.—[ABSTRACT.]

ALMOST all observers state that the virulence of pneumococci derived from different sources, when tested upon animals, is found to vary within great limits; and although Foulerton states that out of 19 cases he found the virulence practically the same in all, I think this can be accounted for by the fact that he did not use very accurate methods of measuring the virulence.

Eyre and I¹ have made a number of observations upon the virulence of pneumococci derived from various sources by inoculation of the rabbit. The virulence of each strain was then tested by injecting measured quantities of the cultivations into other rabbits. The following is the result:

Pneumococcus No. 1.—Derived from the sputum of a case of pneumonia; initial virulence corresponded to a minimal fatal dose of two loops.

Pneumococcus No. 2.—Derived from the sputum of a case of pneumonia; initial virulence corresponded to a minimal fatal dose of one loop.

Pneumococcus No. 3.—Derived from the pus in a case of meningitis complicating pneumonia; initial virulence corresponded to a minimal fatal dose of the whole of a blood-agar cultivation.

Pneumococcus No. 4.—Derived from the saliva of a healthy individual; initial virulence corresponded to a minimal fatal dose of one loop.

Pneumococcus No. 5.—Derived from the lung juice of a case of lobar pneumonia in an infant; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 6.—Derived from the rusty sputum from an atypical case of pneumonia; initial virulence corresponded to 0.01 of a loop.

Pneumococcus No. 7.—Derived from the saliva from a case of carcinoma recti; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 8.—Derived from a case of broncho-pneumonia in an infant; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 9.—Derived from the lung in a case of broncho-pneumonia in an infant; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 10.—Derived from a case of broncho-pneumonia in a child aged $\frac{1}{2}$ year; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 11.—Derived from a case of broncho-pneumonia in an infant; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 12.—Derived from the saliva in a case of interstitial nephritis; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 13.—Derived from the saliva in a case of occupation paralysis; initial virulence corresponded to 0.000001 of a loop.

Eyre, at the annual meeting of the British Medical Association, 1901, recorded five more cases.

Pneumococcus No. 14.—Derived from a case of pericarditis and pleurisy complicating pneumonia in a child aged 8; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 15.—Derived from the pus in the peritoneum of a child aged 3, suffering from pericarditis, peritonitis, meningitis, pleurisy, and fibrosis of the lung; initial virulence corresponded to 0.000001 of a loop.

Pneumococcus No. 16.—Derived from a case of pericarditis in a child aged 15 months; initial virulence corresponded to 0.1 loop.

Pneumococcus No. 17.—Derived from a case of cerebro-spinal meningitis in an infant; initial virulence corresponded to 0.1 loop.

Pneumococcus No. 18.—Derived from a case of empyema following pneumonia; initial virulence corresponded to 2 loops.

The virulence is very easily lost on artificial cultivation; but on blood agar is retained for a much longer period. Eyre and I² found that some strains of the pneumococcus would retain their virulence for 66 days on blood agar, when the tubes were prevented from drying, and were kept at 37.5° C. The virulence can easily be increased by passages through the bodies of rabbits, for example:

Experiment.—A pneumococcus with the initial virulence corresponding to 2 loops has its virulence increased to 0.000001 of a loop after 8 passages through rabbits.

Monti increased the virulence by injecting the products of the bacillus proteus together with the pneumococcus; Pane by simultaneously injecting pneumococci and anthrax bacilli. The diminution of virulence on artificial cultivation can be shown by the following experiment:

A pneumococcus was isolated from the sputum of an atypical case of pneumonia. Its initial virulence was found to be such that an entire blood-agar cultivation injected into the subcutaneous tissue of a rabbit produced fatal septicaemia in two days, whilst one loopful of a cultivation caused a fatal septicaemia in 6 days. After 17 days on ordinary nutrient agar this pneumococcus was transferred to blood agar, when it was found that 6 entire cultivations required 16 days before causing the death of the inoculated animal.

Fraenkel found that one or two days' growth at 42° C. quite removed the virulence. A number of observations have been made upon the virulence of pneumococci at different stages of pneumonia. Patella³ removed pneumococci daily from the blood of the lung, and found a diminution in virulence; and Banti⁴ made similar observations. Weichselbaum⁵ found the cocci taken directly from the lung to be of different virulence when taken at different stages. Welch⁶ found that the most virulent cocci were present in the freshly hepatized lung. Fraenkel also found the cocci most virulent at an early stage.

VARIETIES OF PNEUMOCOCCI

We now have to inquire whether distinct varieties of the pneumococcus exist. Foá⁷ described two distinct varieties of the pneumococcus: the one he named the pneumococcus, producing a serous exudation in the subcutaneous tissue, a moderate number of cocci being found in the blood *post mortem*, and a small and soft spleen. The other variety—the meningococcus—produced no local reaction; the animal died with a large number of cocci in the blood and a hard spleen. He stated that rabbits immunized against the pneumococcus were not immunized against the meningococcus, and vice versa. He was unable to obtain a protective serum from the meningococcus.

Kruse and Pansini⁸ studied a large number of pneumococci obtained from different sources, and they found that strains, when first isolated, differ from one another to a remarkable extent, especially in respect to virulence; that the most virulent strains were most delicate and sensitive in artificial cultivations; and that the less virulent ones were able to grow under conditions in which the virulent ones were unable to flourish. Finally, they came to the conclusion that the one variety could be converted into the other. For instance, the slightly virulent pneumococcus, which would grow in artificial media at a temperature as low as 20° C., could, by passages through animals, be converted into a highly virulent type which would only grow on artificial media at 37° C. On the other hand, a highly virulent variety which would only grow at a temperature of 37° C., could be converted by cultivation into a variety which would grow at 20° C., and during this process the virulence was distinctly lowered.

Eyre and I have made similar observations, and I have generally noted that the pneumococcus which would grow at