Histologically it was shown by Marinesco that the lesions were of an inflammatory nature, and were characterized by adventitial infiltration of the small vessels, particularly the venules, by plasma cells and lymphocytes.

The five following cases occurring about the same time, and during the period of the influenza epidemic in France, are similar in many respects clinically to the case of encephalitis lethargica reported in 1918.

CASE I.

Pte. W.; admitted to hospital on January 5th, 1919. Onset of illness on December 28th, 1918. History of acute onset; malaise, general pains, headache. Temperature about 100° F.

On admission the chief symptoms were mental dullness, slow cerebration, twitchings of both arms and legs. Slight facial paresis on the left side. Pupils dilated; fundi showed engorgement of retinal veins; *tache cérébrale*; no retraction of head, no Kernig's sign, no pyrexia.

January 15th. Condition improving: facial paralysis gone; mental condition better; cerebration quicker.

January 17th. Condition still improving. He was evacuated to the United Kingdom on January 22nd.

CASE II.

Cpl. J.; admitted January 5th, 1919. Onset of illness December 29th, 1918. First complaint was headache, pain in eyes, diplopia and defective vision in left eye; irregular pyrexia.

On admission he was dull mentally, resented being touched, cerebration was very slow; he answered questions slowly but intelligently. The pupils were dilated, but equal, slight internal squint (L.), and left facial paralysis. Slight choked left optic disc. Marked vertical nystagmus present. No history of ear trouble. No specific history obtained. No head retraction; no Kernig's sign; *tache cérébrale* present; deep reflexes increased.

January 7th. Improving. Mentally better; sees better—still has slight diplopia and vertical nystagmus.

January 11th. A few rhonchi in chest. Mild pyrexia otherwise better. Facial paralysis less marked.

January 17th. Facial paralysis gone; great improvement mentally. Temperature normal and lungs clear. Diplopia and vertical nystagmus still persists.

January 24th. Improving; still in hospital.

CASE III.

Pte. R.; admitted January 7th, 1919. Onset of illness January 2nd. Headache and pains in back; eyes tender, complaints of difficulty in seeing; vomited once; no facial paralysis. On admission he was dull mentally, resisted when touched; cerebration was slow; the pupils were dilated and there was some conjunctival suffusion; fundi showed fullness of retinal veins and some obscuring of optic disc margins. Neither retraction of head nor Kernig's sign was present; the knee jerks were exaggerated, and the plantar reflexes of flexor type. Slight facial paresis (L.). He had attacks of intense drowsiness lasting about an hour in which he could not be roused.

January 18th. Condition improving: sees better, headache less; much brighter; temperature normal.

CASE IV.

Pte. J.; admitted January 7th, 1919. Onset of illness December 21st, 1918. Pain in head and back, cough and some abdominal discomfort; complained of tenderness of eyes. On admission there was no pyrexia; he was dull mentally, listless, and not inclined to answer questions. He complained of headache and eye pains; the pupils were widely dilated; the fundi showed fullness of retinal veins, but the optic discs appeared normal; slight facial paresis left side. A few crepitations and moist sounds throughout both lungs.

January 15th. Much brighter mentally; facial paresis gone; pupils still dilated, but no headache or eye pain complained of. Temperature normal.

January 20th. Appears quite normal. Chest clear.

CASE V.

Cpl. R.; admitted January 7th, 1919. Onset of illness December 11th, 1918. He had severe headache and quickly became unconscious. The pupils were unequal, the right larger than the left. Some weakening of left arm and leg. Incontinent.

On admission he was fully conscious, but very dull mentally. He complained of pain in head and both eyes; the pupils were equal and reacted to light and accommodation. The fundi showed marked fullness of retinal veins and obscuring of the margin of the optic discs. There was paresis of the left side of face, and weakening of the grip of the left hand. Deep reflexes were obtained; plantar reflexes of flexor type. Kernig's sign not present; *tache cérébrale* marked. Lumbar puncture: clear fluid obtained under increased pressure. Cell count 200 per c.mm., chiefly lymphocytes. No growth on legum agar after forty-eight hours. Growth on blood smeared agar of a small Gram-negative bacillus. Small Gram-positive diplococci were present also. Leucocyte count, 9,500; lymphocytes and polymorphs in usual proportions.

January 17th. Condition improving; facial paresis still persists. Much brighter mentally; answers questions sensibly, but cerebration still slow.

January 20th. General condition improved, facial paresis almost gone. Much brighter mentally.

January 25th. Still in hospital; improving.

In addition to these, three other cases diagnosed clinically as influenza developed well marked unilateral wrist-drop. Another presented definite peripheral neuritis affecting both arms and legs, while in a fifth case there occurred during the influenzal attack a paralysis of the muscles of the upper parts of the pharynx.

All these cases, as in the first five, were marked by mental lethargy. As all recovered nothing is known regarding the actual anatomical lesion which produced the nervous phenomena, but it seems possible that it did not differ greatly from the lesion found in the cases of encephalitis lethargica.

The question which has still to be decided is, What is the nature of the infection?

A CASE OF HYDROPHOBIA EIGHTEEN MONTHS AFTER INFECTION.

BY

SIR PETER J. FREYER, K.C.B.,
COLONEL I.M.S. (R.).

In 1885, when I was Civil Surgeon of Mussoorie, a case of hydrophobia came under my care which made a great impression upon my mind, partly owing to the distressing circumstances which attended so acute a case, partly because I was constantly at the bedside of the patient, and partly because I was able to ascertain that the bite by a rabid dog to which the disease must be attributed had been inflicted eighteen months before the disease developed. The notes of the case were published in an appendix to the annual report of the Army Medical Department for 1884, but in the circumstances of to-day I feel justified in reproducing the story since that publication is not generally available.

The patient was a lieutenant in a Bengal cavalry regiment, aged 24. I was called to see him at 6.30 p.m. on September 27th, because, as he thought, he had been suffering from fever for the previous two or three days. The temperature was normal, the pulse somewhat rapid and weak; he was bathed in cold perspiration, and seemed nervous and depressed. He stated that he had had fever during the whole of the day, and that he was then in the sweating stage; from the general symptoms I had no reason to suspect otherwise, but there was one symptom I noticed at the time as peculiar. Whilst I was speaking to him he suddenly sat up on the couch on which he was lying and complained of a peculiar sensation as if the upper part of his chest was being compressed. I examined the lungs and heart and found them normal, and concluded that the sensation complained of was due to some cerebro-spinal irritation, such as one sometimes meets with in malarial fevers. He also complained of sleeplessness. He made light of his illness, and stated that it was only in deference to his wife's wish that he had allowed me to be sent for. I prescribed the usual remedies for intermittent fever. He had some quinine pills by him, each containing three grains, and I saw him take two of these. He swallowed them dry, not using any liquid to wash them down. I remember his remarking that he preferred taking them without any fluid. There was then no difficulty in swallowing the pills.

At 5 next morning I had the following note from Mr. T.: "It has just occurred to me that I may have hydrophobia, as I cannot touch water. I was bitten by a mad pup about two and a half years ago at Agra, in the right hand. My right arm pained me a few days ago for no apparent reason." When I saw him half an hour later he was suffering from all the symptoms of well marked hydrophobia. He was lying in bed, bathed in cold clammy perspiration. He was extremely depressed and very restless. The pulse was very weak and rapid. The eyes were sunken, wild, and unnatural. Anxiety, distress, excitement, and terror were depicted on his countenance. He was extremely nervous, disturbed by any sound or movement. The characteristic paroxysms of laryngeal spasm were well marked, and recurred frequently. They set in with a shivering of the body, which gradually increased in intensity, till the whole frame became violently agitated. He sprang suddenly from the lying into the sitting position, with the head bent on the chest, gasping for breath and foaming at the mouth, or he clutched the bed-head rail and struggled violently with the spasm. He spat out viscid mucus, which accumulated in the throat and mouth. The spasms were accompanied by the peculiar dry, hoarse, sonorous cough which is popularly regarded as resembling the barking of a dog, but to which it has no real resemblance. It is produced by the attempt to get rid of the accumulated mucus in the throat by coughing. All the above symptoms gradually increased in intensity till they reached a climax that was distressing to behold; then they gradually subsided, till the patient fell back in bed exhausted, trembling, and bathed in perspiration.

These paroxysms of suffocation were produced by any attempt to swallow either food or drink; even the suggestion

that he should swallow anything was sufficient to bring them on; and he therefore entreated that no attempts to make him swallow medicine should be made, as they would be both useless and injurious. The paroxysms were induced by various external influences, such as the sight or sound of water, any sudden movement of persons in the room, the rustling of a dress, the creaking of boots, the opening or shutting of a door, a current of air, or rays of light. For instance, as the sun rose in the morning the rays of bright light suddenly passed into the room, through a skylight, and this was sufficient to induce a spasm. Again, he heard the noise of the *bhisti* (water carrier) pouring water from his *mushuck* (water bag) outside, and this brought on a violent paroxysm. On one occasion he complained of a desire to pass urine, out when the chamber utensil was brought to him he was unable to micturate, and a paroxysm was induced. He subsequently passed urine involuntarily. The paroxysms of suffocation also came on spontaneously. He could not bear anyone to approach the bed in which he was lying. It was with the greatest difficulty I managed to feel his pulse and administer medicines hypodermically. The fearful paroxysms of suffocation, accompanied, as they were, by the sudden springing up in bed, were sufficient to annerve the strongest man, and would, to the unprofessional mind, give the impression that the patient was a violent maniac. But there was no violence exhibited. The patient was rational till shortly before his death. He spoke sensibly about his affairs, recognized the fact that there was no prospect of his recovery, and met his fate with fortitude. There was no delirium or incoherence. He repeatedly assured me that there was no actual pain, merely the distressing symptoms connected with the disease.

He spoke about his case; he said that two and a half years ago he, amongst others, had been bitten by a pup which subsequently died mad. I afterwards ascertained that the period was one and a half, not two and a half years. The wounds were not cauterized at the time, as the pup was not supposed to be mad. For some time after he had a dread of hydrophobia, but latterly, for months, the idea had completely vanished from his mind. He now remembered that the present attack had commenced three days before with pain in the thumb and forefinger that had been bitten. He said to his wife at the time that he could not hold his pen. This pain passed up the arm and fixed itself in the right shoulder for a time, and there was numbness of the whole right upper limb; this he thought was merely rheumatism. These symptoms were followed by slight fever, with lassitude, weakness, and distaste for food and drink. But it was not till the early morning of the day on which he died that the awful reality flashed through his mind, when he found that he could not swallow fluids.

The paroxysms, already described, at first increased in frequency and violence, each leaving him more and more exhausted; but about noon they began to diminish in frequency and force, and he died of exhaustion at about 5 p.m.

I had the advantage of seeing several medical officers in consultation during the course of the day. They all recognized the hopeless state of the patient. It is only when placed face to face with a case of this dire disease that one recognizes the inutility of any drug to stay its fearful course. During the day hypodermic injections of morphine in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain were administered every two hours or so, and to one dose was added $\frac{1}{4}$ grain of atropine. These had the effect of greatly calming the nervous symptoms and allaying the violence of the spasms.

This case involves several points of interest:

1. The unusual length of the period of incubation. From inquiries I made it was established that he was bitten at Agra in the end of March, 1884. This is clear from the statements of his connexions, and that of an officer of the 29th Native Infantry, to which regiment he then belonged. The period of incubation was therefore one and a half years; the ordinary period of incubation in this disease varies from three to six weeks, and authors distrust the accuracy of statements in cases when the disease is alleged to have occurred at lengthened periods after a bite from a rabid animal has been contracted, and think that the disease is confounded with tetanus and other nervous diseases which have some resemblance to hydrophobia.

2. That this was an undoubted case of hydrophobia, however, there can be no question. I have seen several cases of hydrophobia myself; and all the medical officers that saw this case had no hesitation in at once pronouncing it hydrophobia. Indeed, not one typical symptom of that fearful disease was absent.

3. It is sometimes argued that nervous symptoms resembling those of hydrophobia may be induced by fear after a person has been bitten by a rabid animal, and that these symptoms may actually prove fatal through the shock to the nervous system. No such argument can apply in this instance. The history of the case shows that all fear of hydrophobia supervening had passed away, and that the patient had for three days been suffering from the premonitory symptoms of the disease without any suspicion of its existence entering his mind. It is with a view to make this point clear that I have sketched the

history of the case in a somewhat irregular fashion, and not in accordance with the natural sequence of the symptoms.

4. The most important point, however, connected with the case is, fortunately, of a practical nature, and one to which I desire to call especial attention. The history tends undoubtedly to prove that the poison received from the bite of a rabid animal does not, as in the case of snake poisoning, rapidly invade the system, but remains localized at the seat of injury for an indeterminate period; and, maturing there by some peculiar process, suddenly, after the lapse of days, weeks, months, or years, invades the system. On no other theory can we explain the fact that the first symptom in the actual development of the disease was pain in the seat of the bite. The fact that the patient had no suspicion as to the nature of the pain or of the premonitory symptoms, that he did not recognize the fact that he was suffering from hydrophobia till the symptoms were fully developed, is of very great importance; otherwise it might be argued that the pain was the result of imagination.

The history of this case goes to prove that the poison remains localized for a period which varies in different individuals. If this theory be correct the practical lesson is that free removal or free destruction of the part bitten, by excision or caustics or both, should be had recourse to. For this purpose when a person has been bitten by a rabid animal the fleshy parts around the bite should be excised freely as soon as possible, care being taken that the excision should extend beyond the parts touched by the teeth of the animal. Should a surgeon not be at hand, lunar caustic, a live coal, or a hot iron should be freely applied to the parts so as to destroy the tissues around the bite. In all cases of bites from animals, even when no suspicion of rabies exists, cauterization should be had recourse to. Should these precautions not have been adopted at the time or soon after the bite, the cicatrix and surrounding tissues should be excised subsequently, should it be known or suspected that the animal had been suffering from hydrophobia. I believe there are sufficient reasons to suppose that had such excision been had recourse to in the case under review, even shortly before the active symptoms set in, the disease would never have developed itself. The lessons taught by this and similar cases should be brought home to the multitude. I believe if the measures proposed (which are not new) were more generally adopted, saving of life would result. When once the active symptoms of the disease are developed the result is necessarily fatal.

Grognian Lectures

ON

THE SIGNIFICANCE OF THE CEREBRAL CORTEX.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS OF LONDON, JUNE, 1919.

BY

G. ELLIOT SMITH, M.A., M.D., F.R.C.P., F.R.S.
PROFESSOR OF ANATOMY, UNIVERSITY OF MANCHESTER.

LECTURES II AND III.

(Abstract.)

In the primitive vertebrate the olfactory areas in the cerebral hemisphere, and the visual and general sensory nuclei in the thalamus and the mid-brain, are linked by means of numerous tracts to the hypothalamus, which at first is a relatively large part of the brain. In the pursuit of food, as well as in the animal's other vital activities, this large hypothalamic apparatus is thrown into action as soon as the sense of smell excites a response. By means of numerous descending tracts the hypothalamus exerts a widespread influence upon a variety of visceral and vasomotor functions, some of which stir into activity the glands and other structures directly concerned with the processes of swallowing and digestion, as well as other mechanisms which enable the creature to devote the whole energy of its body to the immediate and vital purpose of the pursuit. And when the consummation of the reaction is attained, and the buccal mucous membrane is