The sterno-hyoid and sterno-thyroid muscles, which are commonly greatly thinned and stretched, can readily be retracted inwards or outwards; there need, however, be no hesitation in dividing them if additional room is required; division of the sterno-mastoid is never necessary. The fascia over the gland is next cleared, the veins in it being at once picked up and ligatured. The gland in its capsule is then isolated and the upper and lower pedicles are identified and freely exposed. This step is essential whether an enucleation or an extirpation is contemplated; if the gland has been thoroughly freed, the enucleation of cysts is easier, and, further, haemorrhage may be readily checked by extroversion of the cavity remaining after enucleation by means of a finger inserted behind the gland.

In an *enucleation* the true capsule of the gland is incised, all bleeding points being clamped and ligatured at once; the gland tissue is then divided over the cyst and the tumour is enucleated by gentle finger dissection. After enucleation, a gauze plug inserted into the cavity for a few minutes will usually check all haemorrhage. Failing this, the manœuvre described above is of value.

Retrosternal cysts often present grave difficulties from their situation and anatomical relations; the best plan to adopt is to clear the upper part of the cyst as far as possible and then to commence the enucleation from below and behind—that is, away from the inferior thyroid and innominate veins. During the necessary manipulations it is advisable to keep the head raised so as to relax the trachea.

In *extirpation* it is most convenient to commence by exposing and ligaturing the vessels entering the upper pole of the gland; the middle thyroid veins are then clamped and divided and the gland is turned downwards and inwards so as to expose the inferior pedicle; this is ligatured *through* thyroid tissue so as to avoid possible injury to the recurrent laryngeal nerve and to leave a small amount of tissue if the whole gland is extirpated.

In cases of hemithyroidectomy I have usually found it best to remove the entire isthmus, any bleeding from the point of division being checked by means of a figure-of-eight stitch of fine catgut. The isthmus may be very adherent to the trachea, especially in old-standing cases or in cases which have been treated by *x* rays, and considerable care is necessary to avoid injury to the trachea, an accident which occurred in one of my cases which had had extensive *x*-ray treatment elsewhere; in this case a small slit was made in the trachea, this was sutured with fine catgut with no untoward result. In complete extirpation each lobe is dealt with separately, the isthmus being left undivided so that the gland may be removed entire.

Drainage is required in all cases so as to avoid the formation of a haematoma, as a certain amount of oozing is inevitable even with the most careful haemostasis. Haematomas, in addition to the prejudicial effect on healing, may cause dangerous respiratory embarrassment from pressure on or kinking of the trachea. Experience of all methods of drainage has convinced me that the best drain is a $\frac{3}{8}$ in. soft walled rubber tube with one side perforation near the end. Drainage is maintained for twenty-four to forty-eight hours.

Suture of the incision should be in layers—that is, platysma and skin separately. The platysma is drawn together by a continuous suture of fine soft catgut, interrupted at the point of drainage; for the skin I now use interrupted fine fishing gut stitches in preference to any other method. At the point of entry of the drainage tube two separate fishing gut stitches are inserted; these are left untied, and traverse the skin and platysma obliquely, so that more of the platysma is taken up than skin; they are tied the day after removal of the tube, and with a little manipulation perfect apposition of the skin edges is possible. Stitches are removed on the fourth day, with the exception of those at the point of drainage, which are left to the sixth day. The majority of the patients were up on the fifth day and left hospital on the eighth to tenth day.

Dressing.—A very abundant gauze dressing should be used in the first instance to soak up any oozing from the wound; over this a pad of wool is applied, and the whole is fixed by means of a double figure-of-eight bandage. In order to prevent the wound being infected by any vomited material the upper edge of the dressing should be sealed down by means of collodion.

After-Treatment.—After the first few hours the most comfortable position for the patient is sitting up supported by a number of pillows. Feeding should be by fluids only for the first two days, after which time ordinary diet may be gradually resumed.

Complications and Sequelae.

Severe *bronchitis* ensued in three of the cases; this delayed convalescence, but left no permanent ill effects—it was in all probability due to the omission of the preliminary atropine injection in at least one case.

Wound haematoma occurred in four cases, fortunately without ill effect other than delayed healing; this was due to the use of too small a drainage tube.

A persistent *mucous fistula*, after the enucleation of a cyst, occurred once. This lasted for three weeks, and was probably due to a mild degree of sepsis.

CASES RESEMBLING ENCEPHALITIS LETHARGICA

OCCURRING DURING THE INFLUENZA EPIDEMIC

BY

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LATE MAJOR R.A.M.C.(T.C.).

THE report on two cases of encephalitis lethargica by Brasher, Caldwell, and Coombe in this JOURNAL (June 14th, p. 733) leads me to place on record the following notes, which I wrote, but did not publish, during the influenza epidemic in France in 1918 and early in 1919, because the cases coincide in many points clinically with those reported by them. My object is to emphasize the possible relation between the two obscure conditions—"encephalitis lethargica" and the "influenza" of last winter.

In that epidemic of influenza many cases presenting cerebral symptoms were recorded. These symptoms usually took the form of drowsiness, delirium, melancholia, and, in rare cases, mania. Cases showing such mental disturbances together with transient paresis of groups of muscles have not, as far as I know, been recorded in association with influenza.

In a report made to the Local Government Board in 1918, on an obscure disease to which the name encephalitis lethargica was given, many cases resembling the ones I detail below were described. They differed, however, from them in certain points — namely: (1) The onset, which was more gradual in my series; (2) the short duration with complete recovery in all of my cases; (3) the ophthalmoplegia with marked ptosis, which was a constant feature in the cases of encephalitis lethargica, was not marked in my series.

Marinesco in a few cases observed a diplococcus in the foci of cerebral inflammation, or in their neighbourhood, but himself admitted that these observations required confirmation.

For purposes of comparison with the cases which I describe below I briefly set down here a few of the symptoms met with in encephalitis lethargica:

Early stage: Slight malaise, with increase in temperature, headache, and drowsiness.

Later stage: Lethargy—sometimes going on to complete mental stupor—paresis of muscles or group of muscles, ophthalmoplegia, ptosis, facial palsy. In some cases third or fifth nerve involvement, together with involvement of peripheral nerves—polyneuritis type.

The main feature in these cases, and the one common to all the cases, was lethargy, and there were always associated with it other nervous manifestations—namely, third nerve involvement, fifth nerve involvement, or polyneuritis.

The close anatomical association of the efferent nerve fibres bringing environmental impulses to the thalamus with the nucleus of the third nerve and its emergent fibres would explain how any lesion in or around that region would produce probably also a cutting off of these environmental impulses and result in a state of stupor, as well as ophthalmoplegia, and other third nerve involvement noted in these cases. The cases of facial paralysis and other nervous manifestations associated with stupor, but not showing any third nerve paralysis, suggest a lesion near but not actually in the nucleus itself, together with other lesions generally in the nervous system—for example, the pons or spinal cord.

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, complains 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 legumen 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

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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 sprung 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