CEREBRAL OEDEMA (EXCESS CEREBRO-SPINAL FLUID):

ITS CAUSATION AND SURGICAL TREATMENT.

BY

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On my return recently from India I read with interest an article by Professor Halliburton on the possible functions of the cerebro-spinal fluid.¹ The article terminated with an appeal for closer co-operation between the physiologist and the neurologist in an attempt to elucidate some of the problems relating to cerebro-spinal fluid. May not the surgeon be invited to this conclave? Surely his experience is likely to be of some value, more especially when he has spent a good many years of his life in the study of injuries and diseases of the brain, and when he has practical experience of problems relating to cerebro-spinal fluid. I have discussed such questions for many years in the wards of the hospital; I have demonstrated on many occasions in the operating theatre the existence of *plus* cerebro-spinal fluid; and now, as the result of Eastern experience, I have further points to bring forward showing the conditions that may lead to excess fluid, the pressure symptoms associated therewith, and the results obtained by surgical treatment.

In discussing this excess of cerebro-spinal fluid, I would utilize the original term applied to a description of the naked eye appearances of brain and membranes when soaked in an excess of fluid, "cerebral oedema." It is both comprehensive and expressive, and it harmonizes with my ideas as to causation. It will therefore be retained throughout.

DEFINITION OF CEREBRAL OEDEMA.

To obviate any possible misconception as to what I mean by cerebral oedema, let me first picture the conditions as seen by the veriest novice in any typical case.

When the dura mater is incised in an operation for subtemporal decompression, carried out for the conditions under discussion, clear cerebro-spinal fluid at once escapes from the subdural space, often spurting out as if under considerable pressure. Under normal conditions there is no fluid in the subdural space, and none escapes when the subdural space is exposed. In the event, however, of considerable excess, the cerebro-spinal fluid which normally occupies the subarachnoid space is forced by the increasing pressure between the cells of the arachnoid membrane into the subdural space. This escape of fluid from the subarachnoid space is not problematical. It can be seen in any typical case of cerebral oedema when the dura mater is incised. The arachnoid presents a "weeping" appearance, the tears collecting into rivulets and running away at the lower part of the exposed area. In relation to the cortical veins, lying in the cerebral sulci and embedded in the subarachnoid tissue, little rivers of fluid can be seen with the naked eye, fluid exuding therefrom and contributing to the general pool that collects below. The brain presents a boggy appearance, and the general surface cortical oedema can be very prettily demonstrated by applying gentle pressure with a sponge, the excess fluid being momentarily squeezed aside, to return as a wave so soon as the pressure is removed. The surface of the brain looks like a very wet sponge.

This excess fluid, seen at the operation, will continue to run away during the sewing up of the wound, and that it continues to run away from the surface of the brain into the subtemporal tissues, both during the healing process and subsequently, is shown by the frequent presence in the temporal region of a fluid swelling; it demonstrates the success of the operation, being the proof that the intracranial pressure has been relieved by the draining away of the fluid excess. That this swelling is a fluid collection can be proved directly both by transillumination and by aspiration, and indirectly by means of lumbar puncture. I have frequently carried out lumbar puncture when it seemed advisable to lessen the degree of temporal swelling, the removal of fluid being followed by considerable diminution in the size and tenseness of the swelling. In the more favourable cases, such as those mentioned in this

paper, the swelling is never very marked, and it gradually recedes till eventually the temporal region lies flush with the surrounding area.

If lumbar puncture be carried out in a case of cerebral oedema, as a preliminary diagnostic measure, the excess fluid is demonstrated, both by the pressure under which it escapes and the quantity that can be collected in a given period of time.

CAUSE.

What is the cause of this cerebro-spinal excess? Professor Halliburton, from physiological experiments, states that there are three groups of substances which promote the flow and increase the pressure of the fluid, independently of those which effect it secondarily by altering the blood pressure—excess of CO₂, volatile anaesthetics, and extract of choroid gland or brain. I leave these physiological causes and turn attention to the cerebro-spinal flow in relation to alterations of blood pressure. Professor Halliburton says that the arterial pressure has but little effect on the amount of secretion. I pin my faith, however, on a very definite relation between the amount of cerebro-spinal fluid present intracranially and the intracranial venous pressure, for the following reasons:

1. It is accepted that the cerebro-spinal fluid is formed either as an active secretion from the choroid plexuses of the lateral, third and fourth ventricles, or by a process of filtration through the choroid capillaries.

2. It is accepted that the greater part of this fluid passes from the ventricular spaces through the foramina in the roof of the fourth ventricle to the cerebral subarachnoid system and spaces.

3. It is accepted that it exists at very much the same pressure as the intracranial venous system, but slightly above the venous pressure.

4. It is accepted that, under normal conditions, it is absorbed, as formed, into the cerebral venous channels, more especially into the lateral lacunae of the superior longitudinal sinus. It would be unwise, in the present state of our knowledge, to make any very definite statement as to whether this is a mere mechanical transmission of one fluid of a lower specific gravity and of higher pressure into another, through a permeable wall, or whether the walls of the veins possess a certain selective action. In all probability the vessels do exercise a selective action, but, under the more ordinary conditions, the absorptive process is probably purely mechanical.

Anyhow, this venous absorption, as I may call it, must be dependent on the healthy condition of the walls of the cerebral venous channels, and on the intracranial venous pressure. Consequently, if for any reason the absorptive capacity of the veins is impaired, either by disease or injury, due absorption cannot take place and cerebro-spinal fluid collects in excess in all possible spaces—in the subarachnoid system, in the ventricles, on the surface of the brain and in the subdural space, producing, as a result, a typical state of cerebral oedema.

For example, if, as the result of heat-stroke, the tunica intima of the cerebral sinuses and veins is impaired, temporarily or permanently, cerebro-spinal fluid will collect in excess. Again, if as the result of some head injury the intracranial venous pressure is considerably raised, so as to be well above the tension of the cerebro-spinal fluid, the normal rate of absorption of cerebro-spinal fluid will be impeded, and it will collect in excess.

There is a third hypothesis, that toxic conditions, acting on the choroid plexuses, lead to an increase in the rate of secretion. Of this, however, there is no considerable evidence.

In the earlier stages of cerebral oedema the excess fluid will escape in the following manner: a small quantity passes down the neural canal, to be withdrawn, perhaps, by lumbar puncture, with some temporary relief to the increasing intracranial pressure; a still smaller quantity permeates through the various emissary channels at the base of the skull, to be absorbed by the extracranial venous channels. As the fluid increases in quantity these means of escape prove inadequate, and it accumulates first in the subarachnoid region, bogs the surface of the brain, and finally exudes through the arachnoid into the subdural space, giving rise to the typical picture of a case of cerebral oedema.

Some further light is thrown on the question by my work in the East. A large number of cases were admitted

to hospital with the diagnosis "heat effects," the great majority coming from Mesopotamia, in batches according to the climatic conditions prevailing in that country. It is obvious that many difficulties were encountered when we endeavoured to classify these cases and group them according to the diseases from which they really suffered. Some proved to be malarial, others were suffering from diseases of the typhoid group, and so on, but there remained a considerable number, running into hundreds, who proved negative to all tests, and concerning whom, therefore, I retained the original diagnosis (heat effects).

SYMPTOMS.

The symptoms from which these men were suffering were as follows:

1. Headache, and General Mental Condition.

Headache, dating from the beginning of the attack, was very severe at first, and then became chronic. When in hospital it was observed that the men were very listless, disinclined to leave the ward, and then only after the heat of the day. They suffered from more or less persistent pains in the head, and were indeed seldom free from headache. They were always worse towards the evening, especially between 5 p.m. and 8 p.m. The pain was usually frontal. During the many exacerbations, the men sometimes retired to bed or to the quieter and darker parts of the ward, disliking the light and their fellows, and burying their heads beneath the bedclothes. They were irritable and despondent. Drugs were of little avail for the relief of the headache, procuring only temporary alleviation at the best.

Operative measures, when proposed, were usually accepted with eagerness, and in some two or three cases the men implored me to do something, at whatever risk, for their relief. The general severity of the headache and the mental effects produced on the patients can be gauged by this ready acceptance of operative measures.

2. Vomiting.

This was present only in the worst cases, and then only in definite relation to the exacerbations of headache. Nausea, of the "sick headache" type, was almost constantly present.

3. Ocular Changes.

Photophobia, more especially during the heat and glare of the day, was a marked feature. True papilloedema was never observed. Every case was examined by our ophthalmologist, but, with the exception of a few in which the retinal vessels appeared rather fuller than usual, there was no definite fundus changes. I anticipated more, in view of the very definite relation between papilloedema and considerable increase of intracranial pressure. However, as papilloedema is not necessarily present in intracranial new growths, even when of considerable size, the absence of any fundus changes in the cases under discussion is quite explicable.

4. Pulse-rate and Blood Pressure.

During the exacerbations of headache the pulse-rate was slowed to 50 or 60, and the systolic blood pressure raised to 140 or 150 mm. Hg—no great rise of blood pressure, but, by taking controls, it was found that the average blood pressure of patients in India was below the normal.

5. Reflexes.

There was a marked tendency to exaggeration of reflexes, both superficial and deep. The men themselves were very shaky and nervous, with tremors of the hands, etc.

6. Temperature.

The temperature was slightly raised as a general rule, with further elevations during the more severe periods of headache, etc.

7. Epileptiform Convulsions.

Epileptiform convulsions occurred in eight of eleven cases here recorded—an unusually high percentage, but capable of explanation. The convulsions were sometimes of the most violent description, each succeeding attack tending to be more severe than the preceding. The men usually knew when to expect the seizure, warned by some excess of headache, curious tinglings, etc. Consciousness was not always lost, the senses perhaps being sometimes

retained throughout a mild attack, and in the early stages of the more severe fits. The duration was variable—from five minutes up to three hours. These men required supervision, and during the convulsions as many as five men were required to hold them down and protect them from self-hurting. Lips and tongue were seldom bitten. Frothing at the mouth was unusual. Struggling was violent, and the face was markedly congested. The blood pressure was raised, and the pulse-rate slowed. After the convulsion was over there was much prostration, sometimes lasting two or three days, associated with headache.

In frequency the fits varied; sometimes a day or two intervened between the attack, or the patient might be free for a week or more.

When the attack lasted two to three hours the patient passed from fit to fit without regaining consciousness. The convulsions were general and it was quite exceptional to see any localizing features.

Several of the cases in which these epileptiform convulsions were observed gave a previous history of brain disturbance of some sort or other—fits, brain fever, head injury, etc. In three cases only were the men normal in every respect. This rather strengthens my views as to the cause of cerebral oedema. If the excess cerebro-spinal fluid is dependent on deficient absorption into the cerebral venous channels, it is only to be expected that antecedent injury or disease would add to the liability to the conditions as pictured. The strain of active service, heat-stroke, shell shock, overwork, etc., would still further impair the rate at which the fluid is absorbed and tend to the accumulation of cerebro-spinal fluid and the advent of definite cerebral oedema.

Interpretation of Symptoms.

All these symptoms, pointing as they do to a general increase of intracranial pressure, together with the *plus* cerebro-spinal pressure (lumbar puncture), and the very satisfactory results obtained by decompression, show that the excess of cerebro-spinal fluid was directly responsible for the development of the symptoms enumerated. Pressure alone might suffice to produce the convulsions, exercised as it was on a highly excitable cortex, the result of heat-stroke, cerebral malaria, etc. It is possible, however, that certain toxic products contained in the fluid may have led to the development of these explosive attacks. Still, by reason of the immediate benefit obtained in every case by simple drainage of excess fluid and the normal characteristics of the fluid collected at the operation, I am inclined strongly to the view that pressure alone sufficed.

TREATMENT.

Many cases presenting these symptoms were admitted to hospital during my fifteen months' service in the East, and seven were submitted to operation. I give a short account of the first case so treated, with an abstract of the six other cases, and, as a control, a brief account of four cases refusing operation.

CASE I.

A R.A.M.C. orderly was admitted to hospital at Amara, Mesopotamia, on June 10th, 1915, suffering from heat-stroke. After recovering he was put on light duty in the wards. There he had several fits which were not controlled by bromides, etc. He was invalided to India; he suffered from two fits at Basra whilst awaiting ship, and one during the voyage. He was admitted to hospital in India on August 13th, 1916, complaining of severe headache, with ready excitement and frequent prostration. His eyesight was said to have been failing for the past few months. The retinal veins were rather dilated and tortuous, especially on the right side. The headache persisted throughout the day, becoming worse in the evening. It never actually left him free, and there were marked exacerbations. All reflexes were *plus*. Tremors of the limbs on exertion. Blood pressure equal to 150 mm. Hg. The pulse-rate tended to be slow during periods of headache. The temperature was never above 100°, and was usually normal.

August 18th. Fit, heralded by abdominal pain and tightness of the chest. Did not lose consciousness; fit lasted fifteen minutes. Twitching of limbs general, then stiffness and general rigidity, followed by severe headache.

August 25th. Severe headache and feeling of a fit coming on. Staved off by walking about.

September 2nd. Severe headache which preceded a violent epileptiform seizure; first clonic spasms of arms, then tonic contraction, spreading to both lower extremities and equal on the two sides. Conscious during the first part of the fit, then senses lost. Fit lasted thirty minutes. Restless afterwards, with severe headache.

September 6th. Numb feelings in both hands, but fit staved off as before.

September 7th. Another fit, more intense and violent than any previous one. Lasted an hour and a quarter. Strugglings very violent, requiring control from four orderlies and convalescent patients. Quite unconscious the whole time. Did not bite tongue. Did not pass urine or motion.

September 14th. No more fits, but headache very violent at times. Getting depressed and miserable. Keeps to bed and sheltered parts of the ward.

September 15th. A still more severe fit, lasting an hour and a half. Very fierce strugglings. Quite unconscious. Followed by headache even more severe than before.

Operation.

Lumbar puncture showed great increase of cerebro-spinal fluid tension and amount. Fluid otherwise normal. Operation advised, and the idea readily accepted by patient, who was willing to undergo any treatment for the relief of headache and fits. I had watched this man daily since his admission and had ordered all available narcotics, with but the slightest momentary effect. For three days previous to operation I kept him under the influence of morphine in order to cut short the possible development of further fits, the result of the operation being obviously more hopeful if performed during a more or less quiescent period. The diagnosis appeared reasonable that, as the result of heat-stroke, the patient was suffering from cerebral oedema, with great excess of cerebro-spinal fluid.

On September 21st I carried out a right-sided subtemporal decompression, choosing that side on the ground that the retinal veins were the more engorged in the right eye. The bone was cut away so as to leave a gap of about two and a half inches in the antero-posterior direction and one and a half inches in the vertical. The dura was opened the full extent by crucial incision. Cerebro-spinal fluid spurted out when the dura was opened, the arachnoid wept copiously, and fluid ran away in rivulets—a typical picture of cerebral oedema.

After-History.

With the exception of some neuralgia and pain in the right face, a common result after subtemporal decompression, due in part to the interference with the temporal muscle, the patient was practically relieved of headache the following day. There was no headache, except of a very mild character, and no fits developed from the day of operation till his arrival in England three months later. The further history of the case is scheduled towards the end of this paper.

Shortly afterwards other cases were admitted to hospital with similar symptoms, some of a mild character, others very severe. The general pressure symptoms were more or less identical, merely varying in degree. There were eleven serious cases, nine with epileptiform convulsions; seven were subjected to operation, and four refused surgical treatment. All cases were watched for an average period of three months, and previous to operation all other known remedies were given full trial.

CASE II.

Fits began at the age of 12. Joined in 1914, but was sent home because of the fits. Rejoined in 1916 and was sent to India. Later, when in Mesopotamia, the fits recurred and became much worse than before, the result, as attributed, of an attack of heat-stroke. The fits were associated with giddiness, headache, and vomiting; they were preceded by attacks of blindness or giddiness, and sometimes lasted forty-five minutes; consciousness was not always lost. When in hospital he had fits on several occasions, three or four days usually intervening. All were very severe and headache was more or less continuous.

Subtemporal decompression, right; much fluid escaped on opening the dura, the arachnoid weeping, etc. No trace of headache, and neither fits nor vomiting subsequently. (Further history below.)

CASE III.

Patient, aged 19, had suffered from fits for many years; infrequent occurrence, but much worse after service in Mesopotamia. Many fits when in hospital, all typically epileptiform. Headache very severe. Subtemporal decompression brought about great relief, and during the two months' residence in hospital after operation the fits became ever less intense and less frequent. (Further history below.)

CASE IV.

Said to have enjoyed excellent health till December, 1916, when, whilst carrying rations during the heat of the day in Mesopotamia, he had a fit, diagnosed as heat-stroke. Mentality poor, headache more or less continuous and very severe at times. Several fits after admission, with loss of consciousness and followed by severe headache. The fits tended to become more severe, the most serious lasted one to two hours, fit following fit, with subsequent prostration. Subtemporal decompression revealed a typical condition of cerebral oedema. After the operation, quite free from headache and fits till leaving hospital. (Further history below.)

CASE V.

Patient had an attack of meningitis when 14 years of age, since when he had suffered from periodic attacks of headache. After service in Mesopotamia he became very much worse. Headaches were very severe, and he declared that his life was not worth living. After several weeks of treatment, he accepted

readily the chance of improvement by operation. Subtemporal decompression showed a condition of typical cerebral oedema and, after the operation, he rapidly improved. Mentality good when discharged, and there were neither headaches nor fits. He said that he was free from headaches and happy, the first time since he was 15 years of age. (Further history below.)

CASE VI.

In this case there was a history of injury to the head some years previously. For some time he had suffered from periodic headaches and vomiting, both of which became much worse in Mesopotamia. Subtemporal decompression showed typical cerebral oedema. He was completely relieved. (Further history below.)

CASE VII.

Quite well in every respect till heat-stroke in Mesopotamia, falling down in the heat of the day in a fit which lasted twenty minutes. A second fit, of three hours' duration, six months later. Subsequently an average of three fits a month. Then invalided to India. During stay in hospital fits became very much more frequent and more severe. Consciousness lost on most occasions. Fits lasted sometimes two to three hours—a succession of fits. Subtemporal decompression showed typical cerebral oedema. No fits afterwards and headache almost entirely relieved. (Further history below.)

As a contrast to these seven cases, I have the notes of four who refused operation. They presented similar symptoms; both fits and headache became more severe and more persistent as the days passed by. I urged operation, guided by my previous experience, but all surgical remedies were refused. The four patients were eventually discharged in a rather pitiable condition.

Reasons for Subtemporal Decompression for Cerebral Oedema.

The drainage of this excess cerebro-spinal fluid, the result of deficient absorption into the cerebral venous system, by means of trephining and dural opening, permits of the escape of the fluid from the confined space in which it is pent up and allows of its coming into contact with new tissues where it can be absorbed. The primary cause (? temporary or permanent changes in the walls of the cerebral veins) is beyond the scope of surgery, but, by the relief of the general pressure symptoms, time and opportunity are afforded for some recovery on the part of the defective venous system, and, if that does not happen, a secondary path of escape has been made for the super-abundant fluid.

All the cases that came to operation, both in India and since, recovered, so that I have not yet had the opportunity of putting my views to the test by obtaining microscopic evidence as to pathological changes in the cerebral veins. And, so far as I can see, there should be no mortality connected with the operation.

Lumbar Puncture.

Lumbar puncture, however frequently repeated, cannot be expected to bring about more than a temporary improvement at the best. This was my experience in several cases. More radical measures are required for the relief of the accumulating fluid.

In addition, there are two points concerned with lumbar puncture which are disconcerting. Firstly, I found that lumbar puncture sometimes made the patients worse, in spite of the fact that a good deal of fluid was withdrawn—one to two test tubes full; and, secondly, lumbar puncture sometimes failed to show any excess at all, but rather the reverse. In one of my last cases, four punctures carried out immediately previous to operation showed a great diminution in the amount of cerebro-spinal fluid; and, in the operation performed immediately afterwards, the opening of the dura showed a most typical condition of cerebral oedema, with very great excess of fluid. Possibly some swelling of the membranes in the region of the fourth ventricle, together with considerable local excess of fluid, closes the normal communication between the cerebral and spinal arachnoid system. Consequently, although lumbar puncture should be carried out in every case, the absence of fluid excess does not by any means negative the diagnosis of cerebral oedema. The general clinical symptoms are a far surer guide.

Indications for Operation.

Operation is indicated in severe and intractable headache, with or without epileptiform convulsions, whether due to heat-stroke, cerebral malaria, shell shock, or neurasthenia. It is obvious that in every case all

ordinary remedies, including prolonged rest in bed (the most important of all), should receive a fair trial, but in view of the injurious effect produced on the cortical cells by prolonged soaking in superfluous fluid, operative measures should not be unduly delayed. I would suggest three months as a fair probationary period.

Region Chosen for Decompression.

The temporal region offers the most suitable site for decompression. The operation is carried out over a silent area of the brain; drainage is conducted at the most dependent part of the brain; the scar is hidden subsequently by the hairy scalp; the bone defect is in a reasonably safe situation, and the operation involves no special risk when carried out by one possessing reasonable skill in head operations. The right or left side is chosen according to the distribution of the headache, but if there is no such localization the right side should be selected to avoid any possibility of disturbing the functions of the motor speech area.

The Opening in the Skull.

The trephine opening should be made low down, immediately above the ear, in the angle between the anterior and posterior branches of the middle meningeal artery. The opening is enlarged mainly in the anterior and posterior directions and downwards towards the base of the cerebral fossa. The bone is cut away so as to leave an aperture not less than two inches in the antero-posterior direction, one and a half in the vertical. The size, however, varies—the more severe the symptoms the larger the opening, and vice versa.

The dura is opened in every case by crucial or other incision. The intradural tension is *plus*, and in consequence the dural incision must be carried out with caution to avoid damage to the underlying cerebral vessels. The temporal muscle, turned down previous to trephining, is sewn into position at the end of the operation. Drainage to the surface should never be employed.

After-Treatment.

The patients should be sat up in bed as soon as possible. I advise small doses of morphine for one or two days following the operation, then bromides for a week or two; they should be gradually discontinued. Patients should be kept quiet in hospital for one month and then sent for prolonged convalescence at some quiet place with open-air treatment and no worries.

Plating the Gap in the Skull.

The situation of the opening, its muscular covering and subsequent protection with the fibrous tissue that develops later, usually render subsequent plating unnecessary. In some cases, however, especially when an extra large opening has been made, protection with a plate may be required. This can be done after the lapse of six months or so.

RESULTS OF SUBTEMPORAL DECOMPRESSION FOR CEREBRAL OEDEMA.

Immediate.

In the seven cases submitted to operation immediate benefit resulted. In some the patients were quite free from headache the day after the operation, the first time for many months. In other cases two or three days elapsed before real improvement occurred. In all cases, however, headache of any severity was a thing of the past. Minor attacks occurred now and then, but nothing compared to the previous severity. As regards the epileptiform convulsions, in spite of the fact that in some cases the fits had been not only numerous and severe but also dated back for some months, and even longer, there were no further fits of any sort (except in Case III) from the time of the operation till the discharge of the patient from hospital, an average period of two months. The mental condition improved in direct ratio; patients previously miserable and depressed became bright and cheerful, eager to get home. All other pressure symptoms disappeared in direct proportion.

Remote.

The later histories of these seven men confirm, with certain modifications, the early results as expressed above. All seven cases arrived home, and communications have

been received carrying on the history of the case down to eighteen months subsequent to the operation, thus giving a reasonable guide as to the result of the measures carried out.

CASE I.—“I am glad to say that I am fairly all right so long as I keep quiet. I often get a head, but nothing like the old days in India. Have only had about four attacks since arriving home.”

CASE II.—“Sometimes I am well in my head and sometimes I am so bad that I cannot do anything. The fits I have not had any more.”

CASE III.—“I am very well sometimes, at others I cannot do anything. I have had no more fits.”

CASE IV.—“I am getting on quite well, and my master is very pleased with my work. I hope you are in the best of health, as it leaves me at present.”

CASE V.—“I am thankful I am able to give you a good report. My head gives me no trouble whatever beyond an occasional headache, which all of us are liable to at times.”

CASE VI.—“I am keeping fairly well. I am getting pains and sickness now and then in dull weather. I get dull headaches, but nothing to worry about.”

CASE VII.—“Up to the time of writing I have had one or two attacks, the last in August of last year, since when I have been in the best of health.”

The first three cases are less satisfactory than Cases IV to VII. This, I believe, is due in the main to the experience gained in the earlier cases, as to points in the operative method, more especially in reference to the size and position of the decompression opening in the bone. But, taking all things into consideration, these results may be regarded as highly satisfactory.

Finally I would add that I have carried out the same operative procedures on three cases recently for chronic headache the result of slight gunshot wounds, being blown up, buried, etc. Two of these men were operated on very recently. In both the early result is eminently satisfactory. One man, the last case, said to me this morning: “My headache is all gone, and I feel a treat.” This man had been blown up and buried, and had suffered from headache for five months.

REMOVAL OF WORM (FILARIA LOA) FROM THE EYE.

BY LIEUT.-COLONEL R. H. ELLIOT, I.M.S.(RET.).

MR. T. W., of the West African Civil Service, was sent to me by Dr. C. W. Daniels on April 2nd, 1918, suffering from a worm under the conjunctiva of the right eye. The patient is highly intelligent and educated. In addition he is an excellent observer, and has taken a very close interest in the evolution of his case, with the result that he has been able to contribute some new and interesting items of information on the subject of this form of filariasis.

History.

He believes that he contracted the disease in Benin City, where he was resident for eleven months (from June, 1909, to May, 1910). He knows of two other Europeans who contracted it whilst there with him, and he considers the district the commonest endemic centre of the disease in West Africa. He has discussed the disease with Sir Patrick Manson, Dr. Daniels, and other well-known authorities, and has taken considerable interest in the flies responsible. He recognized *Chrysops dimidiata*, figured on page 783 of Manson's *Tropical Diseases* (sixth edition), as a very common pest there; he states that this fly is of a yellow colour, and that it settles on its victim “in a very sly and unobtrusive manner, in this resembling the tsetse fly.”

The first signs of infection he exhibited were calabar swellings in the hands and feet; these began ten months after his arrival in Benin district, and have continued off and on up to the present time, but latterly they have only attacked the hands. He describes them as very painful, and complains bitterly of “the tightness of the skin over them.” The first really bad one of these attacks was thirteen months after his arrival in Benin; the previous ones had been quite slight.

His next trouble was the appearance of the worms in and about the eyes. This commenced very shortly after he first noticed the swellings, and has continued off and