suprameatal triangle we can do so from the posterior upper wall of the auditory meatus (Zaufal). This is much more difficult, but there is less risk of wounding the lateral sinus.

We next turn our attention to the soft parts. As the posterior wall of the bony meatus has been removed, the cutaneous meatus now projects into the newly-formed cavity and its posterior part must be cut away or utilized as a flap. Several methods have been suggested: Stacke makes a horizontal incision through the meatus into the concha and then turns the knife and cuts downwards. The lower large flap is then stitched into the lower angle of the post-auricular wound and helps to fill up and cover the cavity. This in my opinion is the most satisfactory method. Korner makes two horizontal incisions; Panse one horizontal, and then a T-shaped vertical incision, upwards and downwards. Ballance makes a horizontal incision in the lower part and then a vertical incision upwards, cutting round the edge of the meatus into the concha. In many cases the healing process is very tedious. Ballance had suggested grafting a large piece of skin from the arm or leg and fixing it over the whole of the large cavity, and two smaller ones on the posterior wall of the ear. He now does not graft the whole of the cavity, but only the upper part. He has discarded gold leaf, and uses small pellets of cotton-wool to keep the grafts in position. He grafts eight or ten days after the operation, and removes the grafts a week later. The objections to this method are, that practically two extra operations are necessary, and the skin grafting is not appreciated by many patients. I do not think that it reduces the time of healing very much, except in cases in which very much bone has been removed.

Another very important question is whether we should close the posterior auricular wound at once, or leave it open, and for how long. I think it advisable in severe or chronic cases not to close the wound at once. I generally put in three stitches and do not tie the middle one, but leave a central opening in the wound; through this and through the external meatus I pack the cavity. It is of great importance to keep the cutaneous meatus well open and dilated. This can be done by plugging, or by inserting a tube, metal or rubber. If we are not careful we get a stricture of the cutaneous meatus, which makes the after-treatment much more difficult. If things do well and there is no rise of temperature, no pus, and no pain, I tie the suture in the posterior wound after five to ten days, and only plug through the meatus. At first I dress the wound daily as long as it is septic, or if there is any rise of temperature. It seems to me to be a most unsurgical proceeding not to dress such a septic wound daily, especially as there is danger of the disease spreading into surrounding vital parts. Hydrogen peroxide is very useful: it cleanses the diseased parts in a most remarkable manner, and is a strong antiseptic. It is essential that the bandage should be as comfortable as possible. I never put it round the neck as, apart from the discomfort, this often causes severe headache.

One of the great difficulties in the after-treatment, is the excessive growth of granulations. These are often brought on by the too-prolonged use of iodoform. After a few days I now plug with aluminium-acetate gauze, and insufflate aristol, isoform, or xeroform, instead of iodoform. In many cases I apply 10 per cent. trichloroacetic acid to the granulations, or 20 per cent. silver nitrate solution.

It is of very great importance that the after-treatment of mastoid cases should be most carefully watched by the surgeon. Many a successful operation has been completely spoiled, and the patient has died, owing to careless or bad after-treatment.

I have no time to discuss all the indications for performing the complete mastoid operation, and will only briefly mention the most important:

1. In all cases of recurrent or chronic mastoiditis with external swelling.
2. Recurrence of granulation masses or caseous matter (cholesteatoma) in the attic or antrum, or persistent otorrhoea, after careful local treatment.
3. Recurrence of pain in the mastoid process, if accompanied by rise of temperature, especially if the pain is in the apex of the mastoid.
4. Fistula over the mastoid process.
5. Cases of chronic otorrhoea with marked stenosis of the auditory meatus.

The points to which I should like to have drawn special attention in this very incomplete paper are:

1. That operations on the mastoid process are often very difficult.
2. That every case is different, and that there is no typical mastoid operation, but the plan and extent of every operation must depend on the area of the diseased bone, and on the topography of the various parts: lateral sinus, mastoid cells, and mastoid antrum.
3. The chisel should be used as little as possible, and the bone forceps and gouge as much as possible.
4. In all cases it is absolutely necessary to open up the attic, remove the posterior wall of the meatus, and carefully explore and open up all the mastoid cells.
5. The great importance of preventing stricture of the cutaneous auditory meatus, and of making a flap or flaps from its posterior wall.
6. The use of skin-grafting when much bone has been removed.
7. The use of hydrogen peroxide for arresting haemorrhage during the operation, and for cleansing the cavity in the after-treatment.
8. The danger of the prolonged use of iodoform in the after-treatment, and the use of isoform or xeroform, and of aluminium acetate gauze.

REFERENCE.

¹ *Lancet*, April 11th, 1903.

THE MICROSCOPIC CHANGES IN THE NERVOUS SYSTEM IN A CASE OF CHRONIC DOURINE,

OR "MAL DE COIT," AND A COMPARISON WITH THOSE
FOUND IN SLEEPING SICKNESS.*

By F. W. MOTT, M.D., F.R.S.

(From the Pathological Laboratory of the London County Asylum,
Claybury.)

DR. LINGARD, of the Imperial Bacteriological Laboratory of India, who has published an interesting monograph on dourine, forwarded for microscopic investigation the nervous tissues of an Arab stallion which acquired the disease by infective coitus; after exhibiting 156 characteristic cutaneous plaques, together with marked symptoms of paraplegia, it died 27½ months after infection.

Dourine is due to a specific form of trypanosome which has the power of penetrating the mucous membrane, affects equines, and is transmitted, like syphilis, by coitus. This is of especial interest, since Schaudinn has demonstrated the *Spirochaeta pallida* of syphilis. It is also of interest because, like some other trypanosome infections—for example, sleeping sickness—it may run a very chronic course, and, as in the case under consideration, more than two years may elapse before a fatal termination.

A comparative examination of the nervous tissues of this disease with those of animals infected with *Trypanosoma gambiense* and with the tissues of human beings dying of sleeping sickness appears to show that chronic trypanosome infection causes a chronic lymphadenitis, followed later by an interstitial inflammation of the connective-tissue structures of the nervous system.

This morbid change is due to the presence of an irritative agent in the lymphatic system, which, in the case of dourine, starts in one seat of primary infection, extends to the inguinal glands, thence presumably by the pelvic lymphatics to the lumbo-sacral plexus, and the posterior roots to the central nervous system; consequently, the lower part of the spinal cord, and especially the root zone of the posterior column, is first and most affected.

Laveran and Mesnil refer to the observations of Marck, who showed the presence of a degeneration of the nerve fibres of the posterior columns, the other parts of the spinal cord being healthy. Marck therefore named dourine infective polyneuritis of the horse.

Dr. Mott was unable to examine the peripheral nerves, because they were not sent, but a very complete examina-

* Abstract of a paper read before the Royal Society on March 8th, 1906, and published in full in the *Proceedings of the Royal Society*, vol. 78, No. B 522.

tion of the central nervous system was made and the following changes recorded:

1. Severe chronic interstitial inflammation of the posterior spinal ganglia—especially severe in the lumbosacral region, but fairly marked in the cervical and dorsal regions also. In the lumbosacral region the inflammation had been so intense as to cause destructive atrophy of the ganglion cells.

2. In the lumbosacral region the posterior roots and their fibre continuations into the posterior columns of the spinal cord had undergone destructive atrophy. This was shown in the spinal cord by a characteristic sclerosis in the posterior columns, especially of the root zones. The lesion resembles, therefore, in some respects locomotor ataxy.

Dr. Mott suggests that the eruption of the cutaneous plaques which came out at successive periods, and which appeared as rings of angio-neurotic oedema, may have been the result of the inflammatory irritation of the posterior spinal ganglia, and not caused, as Lingard and Laveran suppose, by embolism of the cutaneous vessels. In support of this view, he refers to the fact that numerous authorities have shown that herpes zoster in man is caused by an acute inflammation of the posterior spinal ganglia.

Other trophic changes which may be associated with the posterior spinal ganglion lesions may be cited—for example, many of the plaques terminate in a leucoplakia. Towards the end of life in this animal the hoof dropped off. Fractures and dislocations occur, according to Laveran, in this respect bearing a resemblance to tabes dorsalis.

Besides this localized lesion, there was evidence of a general irritation of the central nervous system, manifested by a subpial and septal proliferation of the neuroglia, a chronic interstitial inflammatory change which in the spinal cord was not found limited to the posterior columns.

The changes in the tissues of animals dying after inoculation with *Trypanosoma gambiense*, and the tissues of human sleeping sickness are compared with dourine, and there is evidence to show that chronic trypanosome infection causes a lymphadenitis and a chronic inflammation of the lymphatics of the central nervous system which manifests itself by a chronic interstitial change affecting the lymphatics of the soft membranes, the subpial septa, and the perivascular spaces, without any marked destruction of the neural elements. The neuroglia cell-proliferation is the first manifestation of this chronic interstitial change, and the lymphocyte accumulation and proliferation which is such a pronounced feature of human sleeping sickness, in the light of the observations of chronic trypanosomiasis in animals, may be secondary, and partially the result of these neuroglia changes.

The communication to the Royal Society was illustrated with drawings and photomicrographs. A full description of the histological changes in sleeping sickness and certain trypanosome infections of animals, including experimental sleeping sickness, will be published shortly in the Reports of the Sleeping Sickness Commission.

CASE OF OBSCURE INTRACRANIAL TUMOUR: MENINGEAL SARCOMA WITH EXTENSION TO FOURTH VENTRICLE.

By G. H. GRANT DAVIE, M.B., CH.B.,
WITHINGTON, LANCs.

Mrs. H. J., aged 35 years, married, two children, consulted me on May 8th, complaining of "an aching fullness like rheumatics, only worse," in the occipital region. On March 19th she had an attack of "neuralgia" in the right side of the lower jaw. A few days afterwards she complained of a "rheumatic pain" in the right side of the neck and right shoulder, which "went up and settled in the back of her head, but which was not like an ordinary headache." About the same time she suffered pain in both temporal and inferior parietal regions. These pains continued to increase in severity, with some intermissions, until the middle of April. From this time she could not lie flat in bed, but supported herself in a semi-sitting posture on the right side, for "she felt

she would choke if she lay on her back." Pressure at the back of the neck near the skull gave rise to pain, and sometimes to the choking sensation also. On April 28th she fainted twice owing to the severity of the pain, and because her head "seemed to get off its balance."

The occipital pain always increased in severity between 3 and 4 a.m., after which she could get no more sleep, and made frequent complaint she felt "worn out," as she had had no "proper sleep" since her pains began. Exacerbations of the pain occurred at first once or twice during the day, but latterly more frequently.

On May 8th her symptoms were as follows: She complained of "an aching fullness" in the occipital region and of a track of pain extending forwards to the orbits. This latter pain was of less severity than the occipital, but it became more pronounced when she suddenly rotated the eyeballs, especially in the vertical axis. It was noticed that whilst speaking she placed both hands at intervals under the angles of the jaw, as if to support the head. On suddenly assuming the upright posture she became giddy and placed her hand momentarily over her eyes. She was able to stand erect steadily, with eyes closed, after an initial slight swaying movement. She walked in a straight line and turned with moderate rapidity, preserving her equilibrium. She stated, however, that there were times when she felt she would fall, especially when walking outside and looking at passing objects. She had the sensation of "running forwards on her toes all the time." On one occasion, when suddenly rising, she had vomited, and sometimes "had hard work to keep her food down." In the act of drinking the occipital pain was much increased. The head was therefore thrown back with great care, and rotation of the head horizontally was performed as though she suffered from "stiff neck." She complained of sore throat and some difficulty in swallowing, and also of pain in the lumbar and sacral regions, increased on straightening the back. The tongue was slightly coated white, the fauces injected, the pharynx showing chronic granular inflammation; the appetite was very poor; the bowels sluggish, requiring aperients for every action; the urine was normal; reflexes normal; the temperature normal; the pulse 84.

A week later the occipital pain had extended to the left ear and angle of the jaw, and at 3 a.m., when the pain was most acute, she would call out to be raised to the sitting posture "or she would choke." At a later date she got out of bed when she experienced this acute pain, and knelt with her forehead on the floor, affirming that this attitude gave relief. She described this acute paroxysm of pain as though "a knife was pushed through above the ears to the back of her head." Frequent complaint was made that "she was sure there was something in her head, that this was not an ordinary headache, but if she could only make one understand there was a *something* there."

In order to ascertain exactly the condition of the eyes, the patient was sent to see Dr. Hill Griffith, and the following is his report:

I saw Mrs. J. on May 16th, and found the vision of each eye equal to No. 1 Jaeger and $\frac{1}{8}$; with -0.5 D she got $\frac{1}{8}$. The pupils were normal in size and reacted well to light. The fields of vision (hand test) were normal. The fundi were absolutely normal. The examination was repeated after full dilatation of the pupils by atropine. The vision was $\frac{1}{8}$ with a glass of -0.5 D, and no further improvement could be obtained.

So matters progressed, with decreasing periods of relief, until June 30th, when she got no respite at all from her pain. On July 1st, in the morning, she got out of bed as usual to place her forehead on the floor, and on the second occasion had a violent rigor, "though her body felt warm." The pain, which now extended down the neck to both shoulders, must have been agonizing, for when I saw her at 4.30 p.m. she appeared dazed with it. On this occasion she had a peculiar apprehensive look, and, relaxing hold of her head, she stretched out her arms and cried, "Save me, doctor, I am dying!" Her pulse was then 80, steady, regular, and of good tension; temperature normal. The knee-jerks were exaggerated. The respirations were somewhat "panting."

She complained bitterly of the occipital pain throughout the whole evening, and about 10.30 asked for something to drink. She then appeared to close her eyes to rest, and a few minutes afterwards her attendants found that she was dead.