

tried a very large number I have found none but the method described above which really does so.

About eleven years ago I had an epidemic among my own servants, one of whom had immensely thick and long hair. The infection was discovered late on Saturday night, and on Sunday morning this treatment was carried out. Nothing further was done and no relapse occurred.

About a year ago I was asked by a friend of mine, a physician to one of the London medical schools, if I knew of any certain way of eradicating the pest. His little girl had stayed in a farmhouse and had become infected about eight weeks previously. She had been under the care of a specialist, who had tried more than one method, and at the time that he consulted me his wife was combing the child's hair daily with a fine-toothed comb and cutting out any hair on which she could discover a nit, but up to that time unavailingly. I gave him the method, and he told me later that it was applied only once and the case was permanently cured.

I am also allowed by Dr. A. M. H. Gray, of University College Hospital, to say that he has been using it for some time with complete success, and that even in the out-patient department, where home treatment is liable to be carried out rather carelessly, he does not remember having seen a single relapse.

Lastly, I may mention that there seem to be no disadvantages to the method. I have never seen carboloria result from it, though I should not advise it in very young children as it would be simple to clip off all the hair, and it might be risky to use the carbolic, but with children of 5 years and over it is certainly safe. The hair itself is not in the least damaged, but appears particularly soft and silky after it, a fact which Mr. Cheatle mentioned to me years ago.

If this were made the routine treatment of "dirty heads" in the schools the frequency of the disease would, I think, be greatly diminished.

Harley-street, W.

## Clinical Notes :

### MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

#### A CASE OF METASTATIC ABSCESS OF THE SCLERA FOLLOWED BY PERFORATION AND PROLAPSE OF THE IRIS.

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WHETHER primary suppuration of the sclera ever occurs is held to be doubtful. The meagreness of the literature upon the subject is thus explained. The textbooks generally ignore it. To the absence of blood-vessels in the sclera is probably due its immunity from primary suppurative processes. But, on the other hand, this membrane is fairly freely endowed with blood-vessels around the cornea, and where blood-vessels exist there must also exist the possibility of embolic infection. In panophthalmitis, from whatever cause arising, the sclera may perforate at its weakest point—namely, in the neighbourhood of the limbus. But I have been unable to find any recorded case in which endogenous perforation of the sclera due to sepsis has occurred, save in connexion with a suppurating eyeball. The following case was undoubtedly septic. There is nothing which could account for the ocular signs, apart from the assumption that they were due to a metastatic septic focus. The media remained clear throughout and the vision undisturbed. Moreover, the suddenness of the onset of the attack is in keeping with its embolic origin. Again, the signs of the localised lesion were immediately concurrent with the first complaint made by the patient about the eye. The notes of the case, for which I am mainly indebted to the house surgeon, Mr. A. G. Buckell, are briefly as follows.

The patient, aged 40 years, was under the care of my late colleague, Mr. L. A. Bidwell. The man was admitted into the

West London Hospital on July 2nd, 1912, with symptoms of strangulation of a right inguinal hernia. The bowels had not acted for 36 hours and no flatus had been passed per rectum. The hernia was partially reduced, and on the following morning the bowels acted freely. Further examination of the hernia revealed the presence of a large rounded swelling on the right side of the scrotum, from which the testis was quite separate. The mass was partially moveable, rounded, non-translucent, well-defined below, and with a pedicle running up into the inguinal canal. On the 5th Bassini's operation was performed. The hernia was found to be surrounded by a mass of subperitoneal fat. This mass was dissected out and removed. On the following day a large hæmatoma of the scrotum was observed. Subsequently this increased in size, and two days later was of about the size of a cocoanut and painful, the temperature having risen to 104° F. Later the swelling became tense and tender, and two or three small veins over it spontaneously ruptured. On the 12th, under local anæsthesia, incisions were made into the scrotum, giving exit to much fluid and some blood-clot; temperature 101°. On the 13th the stitches of the hernia wound, which was closed and healthy, were removed. On the 24th large sloughs were coming away from the scrotal wound, which was suppurating freely; temperature 101°.

On July 30th I was asked to see the man. The left eye had suddenly begun to be painful; there was general ciliary and conjunctival injection. In the limbus, in the upper vertical meridian, was a round, yellowish-white swelling resembling in appearance an ordinary phlyctenule seen in phlyctenular conjunctivitis, only much larger. The nature of this swelling was at first doubtful. The cornea was clear, the iris movements were natural, and tension was normal. The inference was formed that in view of the man's septic condition the swelling was probably embolic in origin. Under treatment the eye soon became less painful and the acute injection much diminished. On the other hand, the swelling increased in size. On August 13th it was found to have disappeared and in place of it was a large prolapse of the iris. In other words, perforation of the sclera had occurred. Two days later, the eye being under the influence of cocaine, I excised the prolapse. On August 22nd the man was discharged with his wounds healed and the eye well.

Not much more is required to be said in respect to the ocular signs in this case. The facts, I think, fairly establish the conclusion that the symptoms were due to a metastatic abscess of the sclera. The literature of the subject, so far as I have been able to ascertain, does not contain any recorded case in which a similar perforation of the sclera ensued. I saw the man again on Dec. 5th. The eye was quiet, and the vision was 6/9.

Wimpole-street, W.

#### A CASE OF VERTICAL FRACTURE OF THE PATELLA AND CHARCOT'S DISEASE.

BY A. CLOUSTON RUSSELL, M.B., CH.B. EDIN.,

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THE patient, aged 48, was admitted to the Taunton and Somerset Hospital on May 25th last with the following history. Twelve years ago his right ankle swelled suddenly, but it was quite painless, and did not cause him to stop work. He could not remember injuring the joint at all. After six months this swelling disappeared, and his left knee immediately swelled to a considerable extent. This swelling was also painless, and had no injury to explain its presence. The knee had remained swollen more or less up to the time of admission to hospital. Five years ago the right knee also enlarged suddenly and painlessly without injury. The patient remembered that about a year ago he wrenched the left knee and felt something "give," but he did not take much notice of it and continued his work though the knee was stiff for three or four days. He had had sudden pains in the legs, also "girdle pain" sometimes. There was a specific history.

On examination the right ankle was found to be considerably swollen, the swelling being mainly on the inner side of the joint. There was no redness, heat, or pain. No grating could be elicited, and only slight lateral movement was present. There was marked flat-foot, due evidently to the

softening and consequent stretching of the plantar ligaments and fascia from the effusion. The left knee was 14 inches in circumference, and presented all the characteristics of Charcot's disease, with marked grating in the joint. There was also a vertical fracture of the patella which the patient had never noticed previously to the examination. The outer fragment was one inch larger than the inner. On flexion of the knee the fragments moved outward and inward respectively until two or three fingers could be inserted between them. The main part of the ligamentum patellæ was attached to the outer fragment. The right knee had a circumference of 18 inches, and was rapidly approaching the flail-like condition of the disease. There were very coarse grating, marked atrophy of the bones, and commencing loss of control over the movement of the joint. There was total loss of the knee-jerks, and the Argyll-Robertson pupil was present. The gait was due to the condition of the joints and did not resemble the ataxia of tabes.

I am much indebted to Mr. A. R. Iles for permission to publish the notes of the case.

Taunton.

## Medical Societies.

### ROYAL SOCIETY OF MEDICINE.

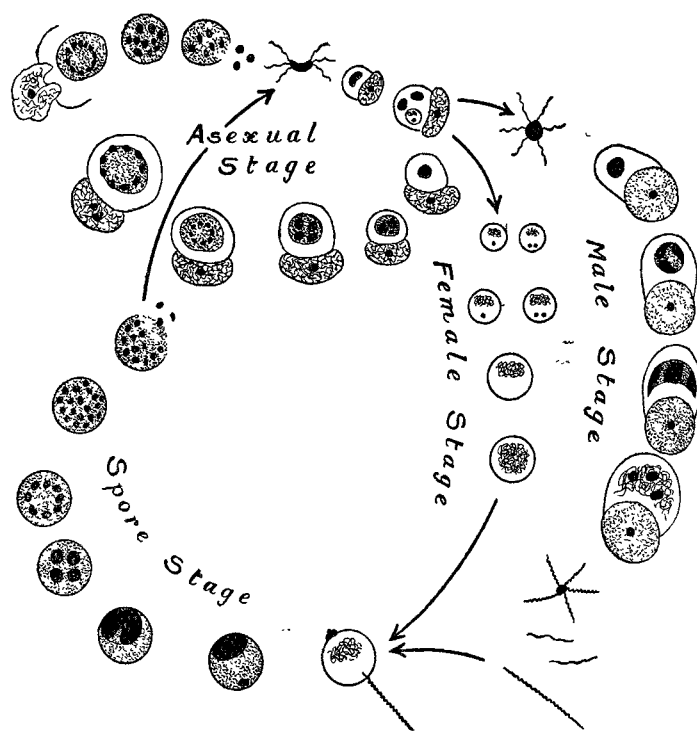
#### PATHOLOGICAL SECTION.

##### *The Complete Life History of the Organism of Syphilis.*

A MEETING of this section was held on Dec. 3rd.

Mr. J. E. R. McDONAGH read a paper on the Complete Life History of the Organism of Syphilis. He said that the sporozoite when examined *in vivo* remained for some time unstained, but later stained very deeply without its motility being thereby impaired. It was seen in two forms: (a) circular, and (b) renal shaped; its size was about  $1\frac{1}{2}$  microns in diameter, and it was actively motile, and occasionally distinctly flagellated. It could be found in syphilitic lesions, in blood withdrawn from the healthy skin around a chancre, and in the general blood stream during general infection. As it could be found in the skin around a chancre when the spirochæta pallida could not be found in the scrapings from the sore, this was of diagnostic importance. The sporozoite then became intracellular; it made a connective tissue cell its host, and when inside it underwent important changes. 1. It steadily increased in size and by a process of budding gave rise to several bodies which later became differentiated into male and female elements. By this time the cell was a sac as all the reserve material had been used up, but the nucleus still remained, although degenerated, to disappear later, then the sac gave way and freed the male and female merozoites. Not all the freed bodies were sexually differentiated, some being able to start the cycle again by seeking a new connective tissue cell. 2. The sporozoite increased in size, but less so than in the previous case. It then divided into two and again into four. The karyosome disappeared after binary fission. The four masses by further division formed a ring which was arranged at the periphery of the body. By this time the host cell was almost entirely degenerated so that the parasite appeared to have become extracellular, but was not truly so till the host cell was no more. In the centre and around the ring other deeply stained bodies appeared until a perfect spore-cyst developed. This was the true asexual stage, and the two phases described represented the schizogony. The sexual generations developed as follows. The male, circular or oval, was actively motile and flagellated. It entered a large mononuclear lymphocyte and became motionless. Then it increased in size, and later three pear-shaped bodies were visible. The male gametocyte steadily developed until a coil formed. In some of the coils deeply stained structures could be seen, which probably corresponded with the pear-shaped bodies. It was also probable that those bodies seen with spirochæta radiating from them like spokes of a wheel were further developments of the same structures. In many specimens stained *in vivo* streptococcal-like chains and many free coccal-

like bodies were visible. Each coccal-like body consisted of a clear ring which contained two deeply staining rods, one above the other, looking like a diplococcus. These bodies were motile. The rods increased in length, and finally a spirochæta developed. These coccal-like chains and bodies, no doubt, developed from the coils. Another body which left the connective tissue cell was also circular, motile, and flagellated, but was a clear body containing a faintly staining chromatin network at its upper pole and one or two deeply staining rods or dots at its lower. It remained extracellular and was doubtless the female gametocyte. It increased to the size of a red blood corpuscle. The dot or dots at the lower pole of the cell were actively motile and probably blepharoplasts, which later left the cell. The chromatin network increased until it filled the centre of the cell. Such a cell was probably the female gamete and ready for fertilisation. Fertilisation occurred thus. The female cell had attached to it a deeply staining body like a cottage loaf. A spirochæta entered. The whole was in active motion. The cottage loaf became extruded, and was probably the polar body or bodies. The spirochæta which entered became deeply stained and more or less rod-shaped. The chromatic network, now located to one pole, finally became transformed into a deeply staining mass, attached in one part to the circumference of the cell. Such a cell was no doubt a zygote. It could be distinguished from the gametocyte by the fact that the cell was stained. Females could multiply by division—parthenogenesis. The changes in the zygote up to the formation of the spore cyst did not differ from those previously described by him. In



Schematic representation of the life history of the organism of syphilis.

early active lesions the sporozoites were best seen. In almost every specimen a coil and spore cyst could be found. In some of the glands he had found little else but coils, as many as five or more in a field. In most specimens the female gametocytes and zygotes were found in greatest number, and it seemed that neither salvarsan nor mercury had any influence upon them. They were equally common in cases free from symptoms as in those with them, and they bore no relation to the result of the Wassermann reaction. The whole life-history of the syphilitic parasite could be followed out in one section from a lymphatic gland which drained the primary sore if removed during the stage of general infection and stained with pyronin and methyl green. Lately he had tried staining films with Giemsa, after fixing while wet in a hot, and then a cold, alcoholic solution of corrosive sublimate, and further treating them before staining with Lugol's solution and sodium thiosulphate. It was laborious, and from a practical point of view could not rival the *in vivo* method of staining with borax methylene blue. He thought he was justified in assigning the organism of