During the treatment I discovered that there was at least one communication with the pharynx. There was no history to be obtained, except the fact that it had been discharging since she was a child, and that the amount of discharge had increased considerably for the past two or three years. An ordinary probe passed without pain through the opening immediately over the origin of the sterno-mastoid upwards for $3\frac{1}{2}$ in., stopping, as it seemed, just above the hyoid bone; on extraction there followed a slight discharge of sero-purulent or sebaceous fluid, but no trace of blood.

Next day I passed a very small gum-elastic urethral catheter, and injected a 1 in 40 phenol solution to wash the track out; some of the solution, the patient said, entered the pharynx and was spat out. She could tell me what the solution was by the taste. The day after I again syringed it out, and passed and left in a silkworm-gut drain; on the third day, after having syringed it out, I twisted together and passed four lengths of No. 2 silkworm gut, having first dipped them in croton oil. I passed them to what I believed to be the upper extremity of the fistula, my object being, of course, to try and destroy the lining mucous membrane; in this it seemed I succeeded to some extent, on account of the copious discharge of pus there was on the dressings the following day. After two more days of antiseptic syringing and draining I passed to the upper extremity of the fistula a silver wire electrode bent on itself, the blunt bent extremity being passed up the fistula, and the two sharp ends of the other extremity fitted into a handle which was connected with the cathode wire. I gradually increased the current from zero up to 5 milliamperes, and immediately reduced it gradually to zero again. I then made the anode the active electrode on the inside of the fistula, and gradually increased from zero up to 10 milliamperes, and reduced slowly to zero again. I then put on a superficial dressing without drainage. The next day, and every day for a week, I applied a flat, flexible tin electrode to the skin surface immediately over the track only, bending the electrode on itself longitudinally, and applying the bent convex narrow border to the skin immediately over the fistula. At each application I first made the cathode the active electrode, increasing the strength of the current from zero to from 15 to 20 milliamperes for a few minutes, and then reducing again to zero, after which I made the anode the active electrode in the same way, but reaching from 30 to 35 milliamperes. Before the application of the alternating currents to the external surface of the fistula, there was a discharge from the exit on applying pressure over the course of the fistula, but after four or five days of the external treatment this discharge could not be produced on any pressure, and at about the end of the week's application the watery external discharge always produced before, on the application of the cathode as the active electrode, ceased externally; but on such application, and at intervals during the day, the patient complained of a bitter fluid discharging into her throat. The applications were continued for another week, up to April 13th, with continued good effect. During the two days previous to April 13th the patient only felt the bitter discharge into the mouth once, and that very slightly. The applications were therefore discontinued on that date.

The cathode, which attracts hydrogen and alkalies, causes, through its irritative and stimulating effect, congestion, softens and liquefies the tissues, causing a watery discharge; on application of the anode, oxygen and acids are attracted, which have a tonic and astringent effect, harden and dry up the tissues, and cause contraction of the passage, so that if this electrode is left in too long it may be impossible to withdraw it without damaging the adjacent parts.

It has been shown¹ that the therapeutic results and chemical changes brought about by a single application of five minutes with a current strength of from 1 to 2 milliamperes so alters muscle structure that evidences of it can be seen for several days after, and so the inter-polar chemical changes set up have caused the cure of this case, possibly assisted by the use of a silver electrode. I do not think that very much therapeutic effect could be attained from the use of a silver electrode, which ionizes with difficulty as compared to metals such as lead, zinc, and copper, although it is, I believe, recognized that on applying the positive pole there is some movement of silver ions into the tissues, causing the liberation of

oxygen and chloride and the formation of insoluble silver chloride.

On April 15th I removed the pharyngeal tonsils, which had been chronically enlarged for some time, causing nasal breathing and night snoring. I ceased attendance on April 30th.

On July 29th I received a report from her father that there was not the slightest sign of the old trouble. I saw the patient on September 15th, and again in January, 1906, and with the exception of very slight redness, no bigger than a pin's head, there was absolutely no sign of the trouble.

The "working-in-the-dark" treatment, as applied in this case, carries with it a certain amount of risk, owing to the proximity of important structures, but by exercising a little care the slight risk is, I think, warranted by the result. The most interesting point about such cases is their rarity and the small number of British or any publications concerning the condition or its treatment.²

As the patient has now been a year without the slightest return of the old trouble, I think I am justified in claiming a cure.

This treatment in place of excision might also be applicable to a patent thyroglossal duct and cyst—a somewhat different state; being a cystic enlargement of a lymphatic in the neck brought about by a fistula or duct from the floor of the mouth or tongue muscles leading to it, and which fistula, I also suggest, is due to an embryological faultiness in the closure of one of the clefts, part of which cleft goes towards the formation of the thyroid gland and isthmus, perhaps the first cleft.

The application of an escharotic without the immediate after-application of galvanism I consider worse than useless, owing to the fact that the irregular destruction of lining membrane and irregular adhesions set up would be more than likely the cause of cystic formations.

REFERENCE AND NOTE.

¹ *Electro-Physiologie*, Weiss, p. 127. ² According to the *Index Medicus*, there were only three publications on this condition in 1903, two in 1904, and two in 1905, and all foreign.

THE INCUBATION PERIOD OF MALTA FEVER.

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THE following case is of interest, for it tells us, within certain limits, the incubation period of Malta fever. A knowledge of this period is of the utmost importance, for it enables us to determine the actual mode of infection from amongst the many possible modes which have been enunciated.

H.M.S. *Lancaster*, having been recently built and newly commissioned, left Portland on June 20th, 1904, for the Mediterranean. She sailed direct for Malta, where she arrived on July 1st. The day after her arrival one of the ship's stokers, who was on his first voyage abroad, was sent to the naval hospital at Valetta suffering from "chancreoid and bubo." He remained in hospital until July 19th, when he returned direct to his ship, cured. The ship left Malta the next day for the Dardanelles, and remained at sea for three weeks. Two days after leaving Malta this man was taken ill with fever.

On the fifth day of his illness he was tested for the serum reaction, which gave a positive result, and the *Micrococcus melitensis* was subsequently separated from his blood. He was invalided home in the following October.

H.M.S. *Lancaster* did not return to Malta until November, 1904, and no other cases of Malta fever occurred on board until January, 1905. It is obvious, therefore, that this man contracted the disease when in the wards of the naval hospital at Malta—that is, between July 2nd and 18th. Supposing that he became infected on the day that he went to hospital, the longest possible period of his incubation is nineteen days, and the shortest two days. The incubation period of Malta fever is somewhere between these two limits, certainly not longer.

Evidence has already been brought forward to show that the incubation period of the disease in human beings, when it has been contracted naturally, is from eight to eleven days.¹ Experimentally, according to the Malta Fever Commission,² the incubation periods of the possible modes of infection are as follows:

A. Infection by Dust Mixed with Cultures of the Micrococcus.—Monkeys fed daily on the artificially-infected dust. Average number of cultures given, twenty-seven. Incubation periods, forty-two to seventy days. Naturally-infected dust given to monkeys failed to produce the disease. Goat fed daily on dust infected with urine containing the micrococcus for thirty-two days ultimately contracted the disease. This experiment has not yet been confirmed.

B. Infection by Food.—Four monkeys fed on cultures for sixty, thirty-eight, thirty-five, thirty-eight days contracted Malta fever. The average number of agar cultures eaten by each of these monkeys was ten. One monkey which only ate seven cultures did not contract the disease. One monkey which ate only two cultures did contract the disease after an incubation period of thirty-two days.

C. Transmission of Malta Fever by Naturally-infected Goat's Milk.—Four monkeys were fed, on the average, three times a day for forty-seven days (mean) before their bloods reacted. It seems probable that animals fed on very large quantities of the micrococci will show an agglutination reaction without infection by the disease; thus Horrocks states that the agglutinins, but not the disease, may be transmitted to the fetus *in utero*. Also a goat fed on naturally-infected milk three times a day from June 27th until October 7th, showed the presence of agglutinins on the latter date, but the micrococcus was never separated from its blood or milk. All these animals were given enormous doses of the *Micrococcus melitensis*.

Four men, all non-immunes, drank some goat's milk, last September, which contained the micrococcus, but they have not up to the present contracted Malta fever.³ From the beginning of July, 1905, all the milk supplied to the troops in Malta, with the exception of one regiment, was boiled, but the Malta fever admissions increased from 67 in July to 77 in September. The regiment which did not boil its milk had the lowest admission-rate.⁴

The milk supplied to the Malta hospitals has recently been carefully sterilized, but the incidence of the disease increased amongst the patients in their wards after the discovery of the micrococcus in the goat's milk.⁵

D. Infection by Inoculation and Mosquitos.—Accidental inoculation in man: incubation period in 3 cases, six days. Experimental inoculation in animals: incubation period, average six days. Natural infection in 4 persons by mosquitos: incubation periods, eight to eleven days.⁶ Accidental infection of a man by a mosquito which had bitten monkeys with Malta fever (the mosquito was found to have the *Micrococcus melitensis* in its stomach): incubation period, eleven days (Horrocks).⁷

The case of Malta fever which occurred on board H.M.S. *Lancaster* can only, according to its incubation period, be classified under the heading D.

REFERENCES.

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MEDITERRANEAN FEVER IN SOUTH AFRICA.

ISOLATION OF THE MICROCOCCUS MELITENSIS.

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DR. P. D. STRACHAN¹ recorded in the *BRITISH MEDICAL JOURNAL* 72 cases of Mediterranean fever which had occurred in his practice in Philippolis, Orange River Colony, and the surrounding districts; 25 reacted with the *M. melitensis* in dilutions of 1 in 10 to 1 in 50.

Unless recently-isolated cultures of this micro-organism are used fallacious results may be obtained. Normal human blood may clump an emulsion of a growth which has had a long saprophytic existence. Konrich² and Cornwall³ have both fallen into error through ignorance of this fact.

Lamb⁴ and I examined the serum of fifty healthy people and of 100 individuals suffering from various ailments. We used emulsions of *M. melitensis* obtained from the spleen of a patient who had succumbed a short time previously. In none of these controls did we observe a complete reaction in a dilution of 1 in 10, nor a trace in 1 in 20. We therefore inferred that complete sedimentation in dilutions of 1 in 10 and over implied the existence of Mediterranean fever, past or present. Forty-four cases in the course of the fever answered to this test. The average maximum dilution in which agglutination was noticeable was 1400. In 68 convalescents the average was

350 if the blood was taken six months, and 250 if taken six to eighteen months after the fever. Our results have been confirmed by the Royal Society's Commission on Mediterranean Fever,⁵ by Nicolle,⁶ and by Lamb.⁷ It is therefore obvious that the serum reaction, if effectually controlled, is a valuable method in the diagnosis of Mediterranean fever, and is only surpassed by the isolation of the *M. melitensis* from the blood of the patient.

Dr. Strachan has sent me capsules of blood from the cases which have occurred in his practice since August last, and of convalescents treated by him previously. The blood of 14 treated during the pyrexia has sedimented an emulsion of the *M. melitensis* in an average dilution of 800. The blood of 18 taken from two to eighteen months after the fever, has reacted in an average dilution of 150. The emulsion was tested with twenty to thirty human serums of various origins in 1 to 10 dilution, and no clumping took place.

The existence of the disease in South Africa has thus been shown clinically by Dr. Strachan, and confirmed by the serum test.⁸ The last link in the chain of evidence to establish it on an irrefutable basis only was wanting—the isolation of the *M. melitensis* from the blood of one of the sufferers. This has been done. The blood of one of the above inoculated on nutrose-agar has given an abundant growth of the *M. melitensis*. This strain resembles the *M. melitensis* derived from the Mediterranean disease in its microscopical, cultural, and agglutinating characteristics.

The origin of the disease in South Africa is interesting. It seems not improbable that it might have been introduced with goats imported into that country. Dr. Strachan has forwarded samples of blood of fifteen of these animals from farms where Malta fever has been prevalent. Two have agglutinated the *M. melitensis* in 20-fold dilutions and over. This indicates that they have been infected.

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¹ P. D. Strachan, *South African Medical Record*, August 15th, 1904; *BRITISH MEDICAL JOURNAL*, July 15th, 1905. ² Konrich, *Zeitschrift f. Hyg.*, Bd. xlv, p. 261, 1904. ³ Cornwall, *Indian Medical Gazette*, 1904, p. 236. ⁴ Lamb and Birt, *Lancet*, September 9th, 1899. ⁵ Reports of the Commission for the Investigation of Mediterranean Fever under the Supervision of an Advisory Committee of the Royal Society, parts i, ii, iii, iv, 1905, 1906. ⁶ Nicolle, *Compt. Rend. de la Soc. de Biol.*, T. 58, 1905, p. 240. ⁷ Lamb, *Scientific Memoirs by Officers of Medical and Sanitary Departments of the Government of India*, No. 22, 1906. ⁸ Birt, *Journal of the Royal Army Medical Corps*, January, 1906.

MEMORANDA:
MEDICAL, SURGICAL, OBSTETRICAL.

ICHTHYOL IN THE TREATMENT OF ANGIO-NEUROTIC OEDEMA.

THE two cases of angio-neurotic oedema reported by Dr. Lowman (*BRITISH MEDICAL JOURNAL*, April 14th, 1906) call attention to a disease designated by Schlessinger hydrops hypostrophos. Of its etiology very little is certain, but that heredity is an important factor is shown by a case reported by Apert and Delille (*Bulletins de la Soc. Méd. des Hôp.*, November, 1904), in which the patient, his father, his only brother, and two other paternal uncles were affected. Professor Osler, too, has recorded an instance in which 22 out of 29 cases were distributed in five generations.

The disease is serious, because it may attack mucous membranes as well as skin, and sudden oedema of the larynx may, therefore, occur and prove rapidly fatal, as in a case recorded by Morris (*American Journal of Med. Sciences*, 1904). Tracheotomy was performed on three separate occasions for asphyxia due to oedema, but the patient succumbed one night from a fourth attack before medical assistance could arrive.

Other cases have from time to time been recorded, notably one complicating enteric fever reported in the *Montreal Medical Journal* in 1898 by Drs. Martin and Gillies. The surgical importance of its visceral crises has been demonstrated by Professor Osler (*American Journal of Medical Sciences*, 1904), and instances given in which laparotomy was performed for angio-neurotic oedema of the intestine, the symptoms closely simulating an acute abdominal lesion. As Dr. Lowman invites suggestions as to treatment, I put forward the claims of ichthyol for what they are worth, having only tried it in the following case: