bedding is in a raised portion of the surface of the endometrium.

6. The solution of the surface epithelium of the uterus is beautifully shown.

7. A parallel is formed by this blastocyst with that seen in the region of recently embedded metastatic areas of chorio-epithelioma, namely, a globular aneurysmal cavity nearby.

8. It appears, from observation, that the human ovum, like that of the guinea-pig, embeds after dissolution of the zona pellucida, and this occurs about seven to eight days after the fertilization of the ovum.

9. From examination, it appears that to facilitate penetration the blastocyst is ovoid when entering the surface endometrium, and only assumes a globular shape after embedding.

I present this specimen in the hope that the embryologists may further study the blastocyst and prove it to have greater value, from an embryological standpoint, than I have been able to demonstrate. It is interesting to compare the anatomical arrangement of the cells in the blastocyst with that reported in Quain's Anatomy of Tarsius Spectrum. (After Hubrecht).

I am grateful to Dr. Geo. L. Streeter,[3] Director of the Department of Embryology, Carnegie Institute, Washington, for explaining to me certain embryological points concerning young human ova; to Mr. Wallace J. Plumpton for the technical study of this specimen.

REFERENCES

1. HARTMAN AND BALL, Annual Report of the Director of the Department of Embryology, Carnegie Institute, Washington, 1931-32, p. 28.
2. BRYCE AND TEACHER, Contributions to the Study of the Early Development and Embedding of the Human Ovum, Macklebone & Sons, Glasgow, 1908.
3 STREETER, Contributions to Embryology No. 92, Pub. 363 of the Carnegie Institute of Washington, p. 31.

RELAPSING FEVER IN NORTH AMERICA, WITH REPORT OF AN OUTBREAK IN BRITISH COLUMBIA

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BRUSSIN and Schapiro[1] conclude from their studies that the various forms of relapsing fever, as found in Africa, Europe, Asia and America, are due to spirochætes developed from a single parent strain which have acquired different antigenic properties owing to different local conditions. Nicolle and Anderson[2] also found that all known strains are closely related. The African form of the disease, which is the most severe and has the highest mortality, apparently does not occur outside that continent. The European form has been prevalent on the Continent and in Ireland since early in the eighteenth century, especially during periods of war and famine. The causative spirillum was first discovered in the blood of a patient in 1868 by Obermeier, and described by him in 1873. Clinically, the disease which is endemic in tropical America and which has appeared in the United States is identical with the European form, although *Sp. novyi,* which is responsible for the American form, shows biological characteristics differing somewhat from those of *Sp. obermeieri.* It is the occurrence of the disease in North America that particularly interests us in this communication.

The first introduction of relapsing fever into this continent appears to have been by Irish immigrants in 1844 at Philadelphia, when Clymer[3] recognized it in 15 cases under his care, all of whom crossed the ocean in the same vessel. The study of another 15 cases was reported in 1848 by A. Dubois. Clymer mentions that it is stated in a report from the Russian government to the British Ambassador at St. Petersburg that the disease was said to exist at New Archangel (now Sitka, Alaska) in 1858. Austin Flint saw the disease in 1850-51, and in 1870 published a lecture based on his experiences with the disease in the wards of the Bellevue Hospital. To his article was appended a statistical report of 103 cases by T. J. Moore. In 1869 the disease appeared in epidemic form, chiefly in New York and Philadelphia, continued throughout that year and the next, gradually disappearing in 1871; a few cases were found in the city of Washington, in Maryland, in New Jersey and in Connecticut; one case was imported into Boston. Osler[4] mentions this epidemic, adding "since when it has not reappeared." In September, 1874, a severe epidemic was observed at Oroville, Calif., among Chinese

labourers. This epidemic resembled both typhus exanthematicus, or ordinary typhus fever, and typhus recurrentis, or relapsing fever. It was probably the latter, but there is no conclusive evidence. Ward reports a case in an Armenian in Worcester, Mass., in 1899. However suggestive clinically, the diagnosis was not confirmed by blood examinations. Carlisle, in reporting 2 cases in May, 1906, was the first in the United States to record having found the spirochæte of relapsing fever in the blood. The origin of one of his cases was undoubtedly tropical America; the other was an accidental infection of a laboratory worker from an inoculated monkey.

At the annual meeting of the Colorado State Medical Society, held on October 5, 1915, Meader[5] presented clinical histories of 5 cases of relapsing fever contracted at Bear Creek Canyon, Col., in 2 of which he found the spirochæte. It is to be noted that these are the first instances of the disease originating among native Americans in North America in which the spirochæte was demonstrated in the blood. In 1918 Waring[6] reported another case from the same region, microscopically confirmed. Since then Briggs[7] recorded in 1922 two cases: a man and his wife in whom the infection was acquired at Polaris, on the Truckee River in California. The next cases were reported in 1930 by Weller and Graham[8]—4 boys of about 16 years of age who contracted the disease while exploring a cave in the Colorado River Valley in central Texas. Bannister[9] reported a case in 1930 in a native Canadian male, a resident of Arizona. Fernan-Nunez, in 1931, reported a case from Wisconsin; this patient, however, flew from Panama only five days prior to the attack.

From the foregoing it will be seen that the disease has been microscopically proved in less than a dozen native Americans.

Epidemiological data on the geographical distribution of this fever, provided by the League of Nations,[10] as well as a search of contemporary and early literature, fail to reveal a single reported case from Canada. The following cases, constituting the first Canadian outbreak, are therefore presented and would tend to show that the disease is already endemic in British Columbia.

All of our patients appear to have contracted the disease while summering on the north-east shore of the lower Arrow Lake region. Only two of the cases were knowingly in contact with each other, and it will be noted that the locations of their different camps are several miles apart. There is no road between Deer Park and Syringa Creek, communications being by boat only. The whole district is sparsely settled, except for a few small farms along the shore.

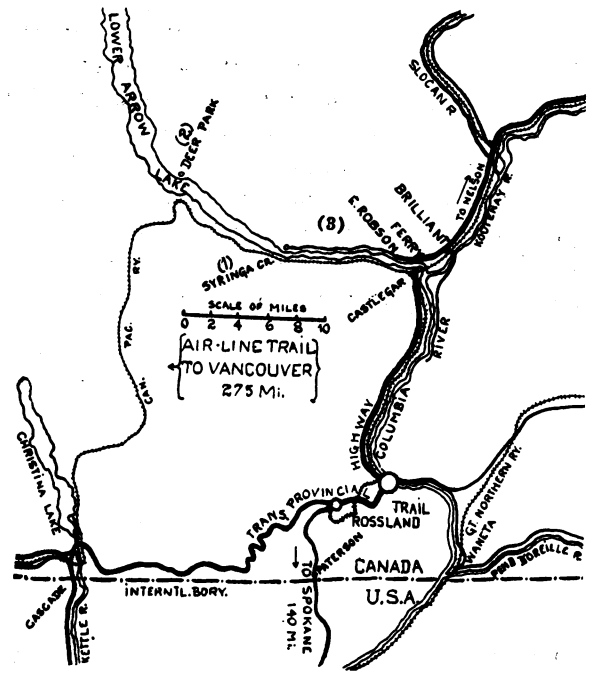

FIG. 1.—(1) Cases 1 and 2, located at Syringa Creek.
(2) Cases 3, 4 and 6, located at Deer Park.
(3) Case 5, located 5 miles west of Castlegar Ferry.

Scattered along the lake between Robson and Syringa Creek are perhaps a hundred summer camps belonging to families from Trail. The district is "off the beaten track" and thus free from transients.

CASE 1

C.B., aged 27, a male, smelterman, born in Ireland, came to Canada in 1926 and had been in Trail ever since. He had spent the week-end of June 25, 1932, at Syringa Creek, fishing. The illness commenced the morning of June 30th, with a chill. He was admitted to the Trail-Tadanac Hospital the same afternoon, complaining of severe frontal headache, chilliness, backache, and pains in the calves and thighs. His temperature was 104°, his pulse 110. At the end of 12 hours the temperature fell to 99°, the fall being accompanied by a drenching sweat. During the next 48 hours the temperature ranged between 99° and 104.6°, rising on two occasions to the latter figure. With each of the two rises there was a rigor, followed by a drenching sweat when the peak was reached. For the remainder of his stay in hospital, which totalled a week, the temperature and pulse were normal. Although he felt rather weak, he complained of no discomfort, and was discharged feeling quite fit. Physical examination revealed nothing abnormal beyond a moderate increase in the size of the spleen.

Exactly one week after the temperature had settled down to normal there was a complete recurrence of his

first attack, which, however, was shorter by about 12 hours. It was in this second attack, while the temperature was rising, that we were able to demonstrate the presence of spirochætes in fresh unstained specimens of blood, and in films stained by Wright's stain, and thus to identify the disease as relapsing fever. Agglutination tests for typhoid, paratyphoid and *Br. abortus* were

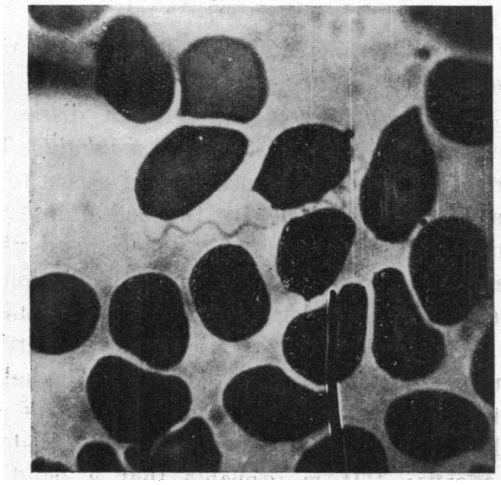

FIG. 2.—Spirochætes in blood, Case 1.

all negative. At the height of the attack the leucocytes numbered 17,000 per c.mm.

The patient refused to have novarsenobenzol administered, and suffered two subsequent attacks, each separated from the previous one by a week, and each one lasting a shorter period than the previous one. After his fourth attack he was becoming quite weakened and decided that if he had another, he would allow us to administer the arsenical. He had no more, however, and regained his former health in a comparatively short time.

CASE 2

J.N., a schoolboy, aged 10, born in Canada, had been staying at Syringa Creek for a month prior to becoming ill. On July 27, 1932, he complained of headache and of feeling cold and shivery. The following day he was considered ill enough to be brought to Trail. He continued to be feverish and to have chilly sensations and profuse sweating; when seen on July 31st his temperature was 104°. Physical examination was negative, except for a tender spleen which could be palpated one inch below the costal margin. On August 1st the temperature was normal, and the patient was apparently well until the second attack, which occurred on August 5th, the temperature becoming normal the next day. Subsequent relapses occurred on August 12th, 19th, 26th, September 2nd, 9th, and 15th. In all of these the original symptoms of headache, chilliness and sweating were exhibited, and the temperature reached between 103° and 104°. None of the attacks, however, lasted twenty-four hours, and in the last three the temperature was normal in less than twelve hours from the onset of symptoms.

When seen for the first time this case was suspected to be one of relapsing fever, and attempts were made at this and subsequent relapses to discover the spirochæte, but were unsuccessful. One intravenous dose of 0.1 grm. of novarsenobenzol given on September 2nd was apparently too small, but no further attacks followed after 0.3 grm. on September 15th.

CASE 3

L.B., a female, aged 15, born in Canada, was employed as a house maid at the summer home of patient C.D. for a month prior to illness. On August 11, 1932, she compained of feeling ill and was sent home to Rossland. On August 13th her symptoms became severe, with backache, nausea, chills, sweating, and a temperature of 105°. She remained in bed until August 18th, when her symptoms and fever subsided. On August 20th she became suddenly ill again with the same symptoms as on August 13th, and again on August 27th another attack took place. These last two attacks, however, lasted only one day each, and no more occurred. She was seen by a physician only during the first attack.

CASE 4

C.D., a male, accountant, aged 30, born in Canada, had been at Deer Park for 10 days prior to becoming ill. On August 14, 1932, he developed a severe headache, aching in the back and limbs, and chilliness, with a temperature of 102.6°. The following day he was feeling somewhat better, but was taken to Rossland, where he was admitted to the Mater Misericordiæ Hospital. On August 16th, the temperature and pulse fell to normal. During this attack the white blood cell count was 10,000. Physical examination was negative, except for slight jaundice. The spleen could not be palpated. The patient was discharged on the 18th, and returned to work on August 22nd.

On August 24th the second attack occurred, with the same symptoms, except that jaundice was absent. It lasted two days. Subsequent attacks occurred on August 28th, September 1st, 4th, 8th, 11th, and 18th, each one being shorter and milder than the previous one. During the attack on September 18th spirochætes were found in the stained blood smear. On September 25th an attack, rivalling in severity the initial one, with a temperature of 102° occurred. Following the injection of 0.3 grm. novarsenobenzol intravenously on September 26th the temperature fell to normal and no more attacks occurred. Agglutination tests for typhoid, paratyphoid, and undulant fever were made at the time of the first attack and again later in the course of the disease, and were negative. Distressing features of this case were the severe headache and insomnia which accompanied the attacks. No definite rigors occurred, but chilliness and sweating were common.

The diagnosis in the next case was not made until a year after the patient had been ill, when the similarity to the foregoing cases was recalled. On purely clinical grounds it seems justifiable to include it as a case of relapsing fever.

CASE 5

J.A.P., a male, aged 24, smelter employee, born in British Columbia, had had no previous illnesses. His illness began while at camp, five miles west of Castlegar ferry, on July 22, 1931, with general malaise, chilliness, and pain under the left costal margin. He was admitted to the Trail-Tadanac Hospital on July 23rd. During the next four days the patient had several severe chills with drenching sweats, headache, nausea and some vomiting, and a foul odour to the breath. His temperature on admission was 103°, pulse 110, and after daily morning remissions the temperature subsided to normal on July 26th. The temperature rose again to 103° on July 29th, with the same chills and sweating, becoming normal again on August 1st. The white blood cell count on July 31st was 5,000. Succeeding attacks occurred on August 3rd, 15th, and 20th, the first lasting two days, and the last two one day each. In each case the temperature reached 103°, and chills and profuse sweating occurred. During the afebrile periods the patient felt comparatively well and free from symptoms. Physical examination was negative, except for an enlarged spleen which was quite tender, and extended three inches below the costal margin. This had become practically normal in size by September 5th. Blood cultures and agglutination reactions for typhoid, para-

typhoid and *Br. abortus* were negative. Several unsuccessful examinations of the blood for malarial parasites were made, but no spirochæte was seen during the search.

The next case dates back to 1930. The patient was seen during his first three attacks by one of our colleagues, who in 1932 recalled the resemblance to the microscopically proven cases.

CASE 6

R.B., a male, aged 23, medical student, the brother-in-law of patient C.D. He spent the week of August 1 to 7, 1930, at Deer Park, at the house occupied in 1932 by Cases 3 and 4, after which he returned to Rossland. On August 13th he took to his bed complaining of mild chills, general malaise, and severe aching pains in the legs and back. His temperature was 102° to 104°. After about four days the temperature fell by crisis to normal, the fall being accompanied by drenching sweats. He felt quite well until August 22nd when the same train of symptoms occurred, with a temperature rise to 105°. He was able to be up on August 27th, and on September 5th the third attack occurred. Two days later he returned to Montreal, where he had three more attacks, making six in all. The interval between attacks was about a week each time, and the later ones were not as severe as the first. After each attack he felt quite well until the temperature rose again. No blood tests of any kind were carried out.

CLINICAL FEATURES

The above cases exhibit the main clinical characteristics of the European type of disease. The incubation period of relapsing fever is given as from one to ten days, most often five to seven; and the onset is usually abrupt. The first febrile period may last from three to ten days, but in most cases it is terminated after a week by an abrupt fall in temperature. After an afebrile interval of from three to eight days a second attack, similar to the first, but shorter and less severe, occurs. At least two more attacks are the rule, but more may take place. The afebrile interval is from five to seven days, and the severity and duration of successive attacks become less. An erythematous eruption sometimes occurs, but this was not noted in any of our cases. The spleen is usually enlarged and tender. Other physical signs may be wanting. Complications are rare, and in otherwise healthy patients the mortality is low, not above 4 per cent.[11]

DIAGNOSIS

The diagnosis is made by finding the actively moving spirochætes in fresh blood specimens during the febrile period, or after staining by Wright's stain in the ordinary manner. In cases where this method fails to reveal the organism white mice or rabbits may be inoculated with suspected blood, the blood of the animal being subsequently examined daily for ten days.

For treatment the intravenous use of novarsphenamine or a substitute organic arsenic preparation is specific. It should however be given during a febrile attack.

VECTORS

The question of the method of spread is a very important one. It is generally recognized that *Spirochæta duttoni* of the African disease is spread by the tick *Ornithodorus moubata.* The European form on the other hand seems to be spread almost entirely by the body louse, although the bedbug appears to take a hand when permitted to do so. Bates, Dunn and St. John[12] have satisfactorily demonstrated that the human tick, *Ornithodorus talaje,* is the transmitting agent of relapsing fever in Panama. The habits of this tick resemble very much those of the bedbug. Waring[6] in commenting on Meader's cases says: "It is probable that a band of gypsies mentioned by Meader brought the disease into Colorado and stopped at the tent where Meader's patients resided, leaving behind infected body lice. Granted this, it follows that the descendants of these pediculi either retained infecting ability from the summer of 1915 until the summer of 1917, a very remote possibility, or other unrecognized cases have come and gone and the said descendants have had opportunity for receiving their infected store of spirochætes." In the cases of the husband and wife reported by Briggs[7] it is stated that they were probably bitten by some suctorial insect, presumably a bedbug or a tick, although this cannot be proved; a great many tramps are found in the area of California mentioned, and there are great opportunities for dissemination of vermin. Graham[13] is inclined to believe that *Ornithodorus turicata* is responsible for his central Texas cases; this tick is found from central Texas southward through Mexico, Central and South America.

As to the manner in which the disease organisms are implanted in the human being, Nogouchi,[14] in an exhaustive consideration of spirochætes says: "In the case of *Spironema recurrentis,* both body lice and bedbugs may be infected by sucking the blood of a patient suffering from the European relapsing fever, but the lice alone can transmit the disease to the next person they bite. Bedbugs are never known to spread the infection by their bites, although

by crushing the infected bugs directly over a minute skin trauma (scratch, etc.), a person may become infected." As regards *Ornithodorus moubata,* Leishman[15] believes that infection proceeds not from the salivary glands, but owing to the fact that the infected excrement voided by the tick while feeding is carried into the wound made by the bite. Leishman has further shown that tick ova may become infected and young ticks be thus born infected. There is no reason to believe that these facts do not hold good for American ticks.

Coming now to our British Columbia cases, we have been able with practical certainty to rule out the louse and bedbug as vectors. There remain ticks, which are notoriously common in this part of the Kootenays. In reply to a request regarding the various species present in British Columbia,, which attack human beings, Arthur Gibson,[16] Dominion Entomologist at Ottawa, writes: "The paralysis tick (or wood tick), *Dermacentor andersoni* Stiles (*venustus* Banks). This is the species that most commonly attacks man in western North America. It is apparently the only tick implicated in causing tick paralysis in man and wild and domestic animals in British Columbia. It is abundant in the Dry Belt areas of British Columbia and in southwestern Alberta. It was reported as unusually abundant in 1932. Its range extends from the United States boundary to at least 100 miles north of Kamloops, and it appears to be most numerous in the Kootenay district. It is only occasionally present in the wet coastal belt of British Columbia. It is considered one of the most injurious ticks in Canada.

"*D. andersoni* is the vector of rocky mountain spotted fever (in the western United States) and also transmits tularæmia. This tick also causes tick paralysis of humans and animals.

"The castor bean tick, *Ixodes ricinus* L., attacks man as well as many wild and domestic animals. Reported from the Pacific Coast region of British Columbia in 1932, causing ulcers in man."

It is noteworthy that the bite of the wood tick is usually painless, and we have encountered cases in which an area of gangrene was forming around the bite, or in which tick paralysis was already developed, without the patient being aware that a live tick was still *in situ.* It is our belief that in the cases which are now reported the wood tick has been the vector. It is confidently expected that more cases will appear this year, and we hope that, if such should happen to be the case, it will be possible definitely to prove or disprove this theory.

SUMMARY

A review is made of the cases of relapsing fever which have been reported in the literature as occurring in North America. Until now no cases have ever been reported from Canada, and the outbreak in British Columbia which is here recorded appears to show that the disease is already endemic in the West Kootenay district. Spirochætes were found in two of the cases reported here.

The problem of transmission is discussed, and the hypothesis advanced that the vector here is the wood tick (*Dermacentor andersoni* Stiles).

We are greatly indebted to our colleagues, Drs. Topliff and Daly, of Rossland, for the histories and findings in cases three and four respectively.

REFERENCES

1. BRUSSIN AND SCHAPIRO, *Arch. f. Schiffs u. Tropen Hyg.*, 1928, **32**: 13.
2. NICOLLE AND ANDERSON, *Bull. Inst. Pasteur.*, 1927, **25**: 657.
3. CLYMER, *New York Med. J.*, 1870, **11**: 55.
4. OSLER, Principles and Practice of Medicine, 8th Ed., Appleton, Phila., 1916, p. 261.
5. MEADER, *Colorado Med.*, 1915, **12**: 265.
6. WARING, *Am. J. M. Sc.*, 1918, **155**: 819.
7. BRIGGS, *J. Am. M. Ass.*, 1922, **79**: 941.
8. WELLER AND GRAHAM, *J. Am. M. Ass.*, 1930, **95**: 1834.
9. BANNISTER, *Southwestern Med.*, 1930, **14**: 581.
10. League of Nations, Monthly Epidemiological Report, 1930, **9**: 481.
11. Nelson Loose-Leaf Medicine, Vol. 2, p. 337F.
12. BATES, DUNN AND ST. JOHN, *Am. J. Trop. Med.*, 1921, **1**: 183.
13. GRAHAM, *Texas St. J. Med.*, 1931, **27**: 226.
14. NOGOUCHI, *J. Lab. & Clin. Med.*, 1917, **2**: 365, 472.
15. LEISHMAN, quoted in Nelson Loose-Leaf Med., Vol. 2, p. 337D.
16. GIBSON, personal communication.

Motion pictures showing how ultra-violet light of certain wave-lengths kills cells were shown before the recent meeting of the American Chemical Society by Dr. Ellice McDonald, Alexander J. Allen, and Rachel Franklin of the University of Pennsylvania. They used cells from the spleen for their experimental material, and the wave-lengths turned on them ranged between 4350 and 2253 Angstrom units. The wave-lengths that were fatal to the cells killed in from fifteen to twenty seconds. The living protoplasm of the cells became greatly agitated, bubbles appeared on the membrane, and as a rule the cells finally burst. The killing effect of the shorter ultra-violet light cannot be equaled by fifteen hours of exposure to strong radiation from radium, nor is the lethal effect of ultra-violet light equaled by twelve to twenty-four hours of exposure to high-voltage x-rays.—*The Diplomate,* 1933, **5**: 129.