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donment of this for stretches of two or three weeks, during which intensive colchicine and salicylate therapy is given "to wash the uric acid out of the tissues". Salicylates and Benemid should not be used together.

Salicylates do reduce the "miscible pool" of uric acid in the tissues. This pool can be of such magnitude that it may take months to bring about an appreciable reduction. It is relatively insoluble, yet continually being added to or subtracted from in the exacerbations and remissions of the disease. Salicylates seem most effective in practice. I have found Benemid satisfactory for maintenance therapy, but drop it in favour of colchicine and salicylates at the first premonitory signs of the general gouty diathesis threatening to break out into an acute localized lesion. I have had no phlebitis since treatment was first initiated, but have had one or two acute lesions in and around the soft tissues of elbow, wrist and knees. These fade out in 24-48 hours on intensive aspirin and colchicine therapy, and resolve in about 10 days. This long-term treatment is essential; discontinuance of therapy as soon as the phlebitis subsides will only bring the treatment into disrepute.

It is not difficult to diagnose an advanced and hopeless case of gout when the classical signs which develop late in the course of the disease are apparent. What seems to be less obvious, and certainly less readily accepted by the profession, is that every gradation of severity occurs from the mildest borderline case to the most advanced. Most of them go undiagnosed. Perhaps recently reported electro-chemical tests, using radioactive nitrogen, will open the way to a better understanding of this disease if they come into common and practical use in hospital laboratories.

In the meantime, it must be realized that the localized pathognomonic lesion, which is simply "a straw in the wind", may be an infrequent occurrence. Until it can be proved otherwise, it would seem reasonable to treat thrombophlebitis as a localized manifestation of gout, on the grounds that *adequate* anti-gout therapy has been so convincingly effective to date.

I acknowledge my indebtedness to Dr. E. M. Wilder and Dr. A. C. DesBrisay for medical care at the time, and for the discussions which helped to formulate this concept.

RECRUDESCENT TYPHUS (BRILL'S DISEASE)

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IN 1898 BRILL[1] observed a disease resembling typhus fever in immigrants from Europe to the United States. During the next few years similar cases were seen in many parts of America. Zinsser in 1934[2] reviewed 538 cases of Brill's disease and advanced the hypothesis that this disease was a recurrent form of typhus fever, the primary attack occurring in an area where epidemic typhus was prevalent. Zinsser postulated a latent period during which the typhus organisms persisted in the tissues, the factor responsible for a recurrence of the active disease being unknown.

The clinical picture of recrudescent typhus is similar to that of epidemic typhus, although the course of the disease is generally less severe. The rash and the serum agglutinins for *Proteus* OX 19 associated with epidemic typhus are both usually absent in recrudescent typhus.

Mrs. T.M., 51, Polish Ukrainian, was admitted to the Winnipeg General Hospital on November 6, 1953, with the following history.

During the summer of 1953 she complained of a cough, excessive lacrimation and a choking feeling in her throat. Her physician made a tentative diagnosis of goitre, and iodine therapy was instituted. The patient felt reasonably well until October 1, 1953, when there was an abrupt onset of fever and headache. The headache was situated at the vertex and was sharp and continuous; there was no frontal or occipital aching. On November 2 and 3 she vomited and had occasional chills. On November 4 she was given an injection of 400,000 units of penicillin. This treatment had no effect on the fever, but there were no further chills. On admission to the hospital the patient complained of feeling feverish and of an extremely severe headache.

In 1928 an ovarian cyst and the appendix had been removed. In 1951 a hysterectomy had been performed. The patient had had a previous episode of dermatitis.

Physical examination.—The patient did not appear unduly ill although her temperature was 104° F., pulse rate 120 and respirations 20. She was complaining bitterly of the headache. There was no neck rigidity. With the exception of a palpable isthmus of her thyroid, the remainder of the physical examination was essentially negative. There was no rash and the spleen was not palpable at any time during the illness.

On November 6, the leukocyte count was 9,000/c.mm., with 80% mature neutrophils, 4% young neutrophils, 13% lymphocytes, 1% monocytes and 2% degenerated cells. Hæmoglobin value was 90%; red cell count 4.8 million/c.mm. E.S.R. (Westergren) was 67 mm. in one hour. A lumbar puncture was performed with

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TABLE I.

AGGLUTINATION REACTIONS

Date of blood collection	*Nov. 9, 1953*	*Nov. 23, 1953*	*Dec. 22, 1953*	*Nov. 22, 1954*
Proteus OX 19 agglutination	1/320			1/20
Complement-fixation test:				
Epidemic and murine combined typhus antigen		1/256	1/64	1/8
Epidemic typhus antigen		1/512	1/256	1/16
Murine typhus antigen		1/32		0
Blood samples were later sent to Dr. Michael Sigel, Communicable Disease Center, Montgomery, Alabama, for complement fixation tests for confirmation:				
Epidemic typhus antigen	1/1024	1/1024		
Murine typhus antigen	1/256	1/64 or 1/128		

an initial pressure of 240 mm. of water and a final pressure of 200 mm. Five c.c. of clear fluid was removed which had a total protein content of 10 mg. %: the fluid contained 17 leukocytes and 5 lymphocytes per c.mm. Colloidal gold test was negative. Urinalysis was normal. Two blood cultures were reported negative. The roentgenogram of her chest was normal. Blood agglutinations are reported in Table I. On November 9 the leukocyte count had increased to 16,000, with a differential count of 60% mature neutrophils and 18% young neutrophils showing toxic granulation, 13% lymphocytes, 8% monocytes and 1% degenerated cells. A second urinalysis revealed a trace of albumin.

In the absence of a definitive diagnosis, no antibiotics were given. On November 6, 7 and 8, the patient's temperature rose from 104 to 105° F., subsiding to 99° F. on November 9; the patient then remained afebrile until she was discharged from the hospital on November 14. On November 23, when a provisional diagnosis of recrudescent typhus had been made, the patient returned for further questioning. She stated that in 1918 or 1919, when in the Polish Ukraine, she had suffered an attack of the "flu"; the members of her family were also sick and the woman nursed them in spite of her own disability. Later she became dangerously ill for a period of three weeks and had been informed that she was near death during this period. In the course of this illness all the hair of her head fell out. The patient emigrated to Canada in 1921 and until the present attack had suffered no illness with symptoms resembling those of 1918-1919.

DISCUSSION

A rickettsial infection was suspected in this case when the *Proteus* OX 19 agglutination test was positive; this finding is unusual in recrudescent typhus. The complement-fixing antibodies had apparently reached a peak in the serum by the time the first blood sample was collected, 10 days after the onset of illness. As shown in this case, specific serum antibodies increase much more rapidly in recrudescent typhus than in the primary attack of epidemic typhus. The third sample, collected 52 days after the onset of the disease, showed a slight decline in antibody content compared to the second one. The fourth sample, collected approximately one year after the illness, showed a marked decrease in the antibody titre.

The complement fixation test with epidemic typhus antigen showed the highest titre of antibodies; using the murine typhus antigen, a low but definite titre of antibodies was obtained. These results illustrate the cross-reaction that other workers have observed between these two antigens.[3]

A case of recrudescent typhus in Canada is primarily important as a possible focus of infection from which an epidemic of classic epidemic typhus could originate. This potential hazard to the health of the public appears to be the most significant aspect of cases of recrudescent typhus in this country. In view of the large number of immigrants from Central Europe, where epidemic typhus has been rampant, it is surprising that more cases of the disease have not been reported in Canada. The possibility exists that some undiagnosed fevers in immigrants may be recrudescent typhus.

Epidemic typhus flourishes in countries where a cold climate and poor living conditions predispose to an increase of louse infestation. Certainly the winter season in Manitoba is cold and, although living conditions are generally good, every large city possesses a section where overcrowding supplies the necessary requirements for the spread of epidemic typhus.

SUMMARY

1. A case of recrudescent typhus has been reported.

2. The diagnostic procedures are reviewed.

We wish to express our appreciation to Dr. G. P. Fahrni for permission to publish this case.

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