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point. On November 30, when the vitamin E ointment was increased to full strength, the ulcer base appeared quite healthy with good granulations, and the sides were beginning to fill in with healthy-looking skin.

On December 15, the ulcer measured only 5.5 x 1.4 cm., with a depth of only 0.1 cm. Granulations were all healthy and there was no evidence of any degenerating tissue. This case again presented the problem of management of a woman who would not help herself and spent most of her time either lying or sitting down, with the added factor of frequent urinary incontinence.

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WESTERN EQUINE ENCEPHALOMYELITIS: REPORT OF A CASE IN MONTREAL

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WESTERN equine encephalomyelitis (W.E.E.) has been recognized as occurring in humans since 1938 when Beatrice Howitt of the University of California isolated the virus from the brain of a 20-month-old boy who had died on the fifth day of his acute illness.[16] An encephalitis had been observed in horses and mules for many years in the U.S.A. but it was not until the summers of 1930 and 1931, during two epidemics in California, that a virus was demonstrated and isolated as the etiological agent through the studies of Meyer, Hering and Howitt. The possibility of this virus causing disease in humans was suggested at that time by Meyer.[15]

In Canada, the first recognized epizootic in horses occurred in Saskatchewan in the summer of 1935. This was followed by more severe outbreaks in 1937 and 1938 which included the Province of Manitoba. In the wake of this equine infection a number of humans developed an unidentified disease of the central nervous system which at first was thought to be non-paralytic poliomyelitis. This was later established as W.E.E. from two human cases by Fulton and Burton in 1939.[3]

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The most extensive human epidemic occurred in 1941, involving the Provinces of Saskatchewan and Manitoba, and the states of North and South Dakota, Minnesota, Montana and Nebraska. There was a milder epidemic in 1947 and since that time the whole west has been plagued with endemic infection. As there have been no reports of human W.E.E. in Canada, east of Manitoba, it was thought important to report an atypical case of this disease occurring in Montreal.

D.A., a 20-month-old male infant, had been attending the allergy clinic of the Montreal Children's Hospital for treatment of recurrent attacks of asthma for six months previous to the present illness, which began on September 17, 1955. On that day he developed a hoarse voice, a dry hacking cough and dyspnœa. At 2 a.m. on September 20, because of increasing dyspnœa and restlessness he was taken to the out-patient department where on examination he was thought to have an acute laryngitis or epiglottitis with an allergic component. He was treated with intramuscular chloramphenicol 500 mg., a subcutaneous injection of 0.5 c.c. of adrenalin (epinephrine) 1 in 1000, ½ of a 0.25 g. aminophylline rectal suppository to be continued q.6.h., Franol (ephedrine, luminal, theophylline) ½ tablet q.i.d. and Elixir of Pyribenzamine (tripelennamine) 10 mg. q.i.d. By 10 a.m. the same day on his return to the clinic, he was much improved but by 10 p.m. his respiratory rate was again very rapid with dyspnœa and pallor; he was again brought back to the hospital and admitted the night of September 20. When seen on the ward he was very pale, showed marked respiratory difficulty with supraclavicular, intercostal and subcostal indrawing, and was thrashing about his bed making purposeless movements. His temperature was 102° F., his heart rate was 200 per minute and his respiratory rate was 60. The remainder of the physical examination was done with considerable difficulty and was non-contributory. He was placed in an oxygen croupette, given intramuscular chloramphenicol and streptomycin, and the aminophylline and Pyribenzamine were to be continued. The department of otolaryngology was consulted and on their advice emergency tracheotomy was postponed. Eight hours after admission his respiratory distress had improved but his peculiar maniacal behaviour persisted. He had no other abnormal signs referable to the central nervous system apart from an equivocal nuchal rigidity. The optic discs appeared normal. The nurses noted on two or three occasions that he had tremors of his upper extremities. Sedation with phenobarbital was unsuccessful but was finally achieved on his third hospital day with chlorpromazine. His temperature ranged between 101° and 103° F. until September 25, when it remained at 100° F. for 48 hours before it subsided to normal on September 29. He showed definite signs of improvement by the end of the week, and by his tenth hospital day he was playing like any healthy child of comparable age.

Laboratory investigation.—Hæmoglobin value 8.6 g. %, white cell count 11,200 with 6200 neutrophils, 4400 lymphocytes and 800 monocytes. There was no stippling of the red cells. Urinalysis was normal. Intradermal tuberculin and blood Wassermann tests were negative. Blood calcium, potassium, sodium, chlorides, CO_2 combining power, sugar and non-protein nitrogen were within normal limits on September 22. Cerebrospinal fluid (CSF) samples obtained on September 21 and 26 were clear and colourless with only two cells and a negative Pandy test in each case. The protein, sugar and chlorides of the CSF of September 26 were within the normal range. Roentgenological examination of the

long bones revealed no lead line or any abnormality. The chest and skull radiographs were not abnormal. A blood culture taken on September 21 was negative. A very light growth of *Staphylococcus pyogenes* was obtained from the nose culture. An electroencephalogram taken on September 22, only 24 hours after phenobarbital was discontinued, was reported as showing "no definite abnormality".

One of us (V.P.) carried out virus antibody studies on this child. The following are the results of the complement-fixation test:

Days after admission to hospital	*Titres of complement fixation antibodies for W.E.E. in the serum*
6	0
14	1:4
23	1:8
25	1:32
1 year	1:8

All of the serum samples gave a negative complement-fixing reaction with the following viral antigens: lymphocytic choriomeningitis, Eastern equine encephalitis, St. Louis encephalitis, mumps and Japanese B encephalitis. The tests for neutralizing antibodies were positive in all serum samples, but the virus could not be isolated from the samples of serum, CSF, stool, nose and throat swabs sent on the sixth day of illness.

There was no family history suggestive of an encephalitic syndrome, no history of contact with horses or fowl, and the family had not been out of Montreal since the birth of the child. Serological tests on the father and an older sibling, aged five years, gave negative results. The mother was found to have complement-fixation antibodies for W.E.E. in a titre of 1:16 but she gave no history of illness. One year later the titre was 1:4. Local veterinarians, when contacted, reported that they had not seen or heard of any cases of this disease in any animals.

The child was seen at the neurology out-patient clinic, one month, six months, and one year after discharge. During the first two visits, the mother complained of marked personality changes in the child since discharge, e.g., fearfulness, destructiveness and temper tantrums. By the last visit, she reported definite improvement. At all visits, the neurological examination showed no abnormalities. However, an electroencephalogram taken on his last visit showed "an area of dysfunction—potentially epileptogenic in the right occipital area".

A psychometric analysis was done on his last visit as well, and the psychologist reported the child to be functioning at borderline mental deficiency. However, we have no pre-illness tests for comparison, and, furthermore, psychological tests at this age must be interpreted with great care.

An attempt was made to rule out the possibility of aminophylline intoxication, particularly in view of the similarities of this case to the cases of White and Daeschner, recently reported.[17] The child had had aminophylline suppositories on three known occasions before this illness without any unusual response. He had received 3¾ grains of the suppository over 12 hours before this admission and 1¾ grains immediately after admission. We gave the child 3¾ grains of suppository as a test on his last follow-up visit and no untoward signs occurred. No definite conclusions can be drawn from this test, but it is highly unlikely that the serological changes that occurred could be due to this idiosyncrasy, or that any type of anamnestic stimulation (virus or bacterial) could have occurred.[13]

DISCUSSION

A. *Clinical*

W.E.E. has such varied clinical manifestations that the differentiation of it from non-paralytic poliomyelitis and the other acute encephalitides is impossible without serological tests. It has the same seasonal incidence as poliomyelitis, prevailing mostly from mid-July to mid-September. The highest age incidence is reported to be in adult males 20 to 50 years of age, working outdoors. However, in the Manitoba epidemic of 1941, infants of under one year of age had a high incidence. The vector is thought to be a mosquito, a wild bird mite, chicken mite or wood tick, although proof of this is lacking. Chickens and other birds are suspected of being reservoirs of the virus.

The incubation period is 5-10 days with extremes of 4 and 21 days. The initial symptoms are mild, consisting of headache, fever, drowsiness and gastro-intestinal disturbances. As these symptoms are subsiding, in 1 or 2 days, there is a sudden elevation of temperature with acute neurological signs predominating. The febrile period lasts 7 to 10 days. Paralysis is found in 15% of the cases. Atypical types such as (1) abortive forms, consisting of fever and headache, and (2) inapparent forms, with little or no clinical disturbances but with definite serological changes, are not uncommon. A mild epidemic in Saskatchewan a few years ago masqueraded as an influenza infection.[3] Most of the cases reported in infants were explosive in their onset and portrayed a fairly complete picture of encephalomyelitis.

The onset of the infection in our patient was acute but the initial symptoms were those of an acute laryngo-tracheo-bronchitis with marked restlessness. Only after the respiratory difficulty had abated with no improvement in his agitated behaviour was the diagnosis of some type of encephalitis entertained. The cerebrospinal fluid, as mentioned above, was normal and is reported normal in rare cases. The more common finding is a pleocytosis (10-400) with polymorphonuclear leukocytes predominating early in the disease and later mononuclear cells; the protein content is usually elevated and the sugar content slightly decreased or normal.

The mortality rate varies from 7 to 20% with an average of 10%. The reports regarding sequelæ differ. Adamson and Dubo, reporting mostly on adult males 16% of whom were over 60 years of age, have claimed that most patients recover completely.[1] Medovy found that of 17 infants with W.E.E., 12 made a complete recovery and five had sequelæ.[7] Everyone who has fol-

lowed up cases of W.E.E. over a period of time has found a definite number of sequelæ attributable to the disease, e.g. (1) inability to use one side as well as the other, (2) focal or generalized convulsions, (3) rigidity, (4) speech difficulty, (5) retarded mental development.

Our patient was thought to have average mental development before the onset of the illness. No actual testing was done then, so that the fact that he is now functioning at "borderline mental deficiency" is difficult to assess, but combined with the EEG findings of "an area of dysfunction —potentially epileptogenic in the right occipital area", it may have some significance.

B. Serological

The diagnosis of W.E.E. can be confirmed only by isolation of the virus or serological examination. A suggestive clinical picture is conclusive when accompanied by one of the following:[18]

1. Virus isolated from the central nervous system or blood of the patient. Virus can most frequently be obtained from grey matter of patients who have died within the first five days after onset and when autopsy is performed within two or three hours after death. Other tissues, blood or cerebrospinal fluid rarely if ever give positive results.

2. The demonstration of a rising titre of neutralizing or complement-fixation (C.F.) antibodies. The neutralizing antibodies reach high titres in one week and stay at these levels for about two years, and gradually disappear. They have been found as late as 12 years after the initial infection.[4] Complement-fixation antibodies begin to appear at the end of the first week; their titre rises slowly and begins to subside again after six to eight weeks. Usually they are detectable up to 12 to 14 months.[11]

3. The presence of low titre of complement-fixing (C.F.) antibodies, in the absence of the antibodies against other encephalitides, may have considerable significance in diagnostic studies. In general, a four-fold rise of C.F. antibodies is considered significant.

In a few instances antibodies of the W.E.E. have been detected in individuals in Northern Ontario and in people in Ontario who have come from western Canada or the U.S.A. The Epidemiology Division of the Department of National Health and Welfare has reported: "To the best of our knowledge, the virus has never been isolated in Quebec and there have been no cases of disease caused by the Western variety of the virus in Eastern Canada before."[14] "It has been reported that this year, investigators in New Jersey have isolated antibodies to the W.E.E. virus in a pheasant."[14] Mitchell and Pullin[12] collected, at random, sera in 1942 for pre-inoculation assessment, and detected an antibody incidence of 18.95% in 1013 cases in Manitoba, 1.98 in Ontario and none in 50 cases tested in Quebec. Pavilanis[13] studied the sera from 100 patients in a mental hospital in Montreal and found no antibodies for W.E.E. In checking through the records of four years of operation of the Virology Laboratory of the Institute of Microbiology in Montreal, he had only one case in 1952 which showed a low titre for antibodies of W.E.E.

CONCLUSION

The case of a 20-month-old infant with a clinical picture resembling acute laryngo-tracheobronchitis but with significant rising titres of antibodies of W.E.E. has been presented. This appears to be the first reported case of this disease east of Manitoba. The literature on W.E.E. has been reviewed and the diagnosis discussed. In summary, we suggest that all patients with symptoms suggestive of encephalomyelitis and those with neurological disturbances of undetermined etiology should have serological testing for neurotropic viruses.

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ALCOHOLISM

In Canada, alcoholic employees lose about 19 working days per year as a result of this illness—more than twice the absenteeism of non-alcoholic employees.