includes almost every neurological symptom and sign occurring in hypertension. Headache, dizziness, convulsions. dementia, attacks of vasospasm and small strokes are often referred to under the term. A classification into acute and chronic hypertensive encephalopathy can be convenient when the former is used for the acute often terminal syndrome comprised of headache, vomiting, papillœdema, convulsions, stupor and coma, associated with brain œdema, while chronic hypertensive encephalopathy is used to refer to the state following upon repeated small strokes and attacks of so-called vasospasm. Headache and dizziness do not as a rule connote disease of the brain. Typical vertigo even of sudden onset as an isolated symptom in the hypertensive patient rarely indicates a stroke, being generally of labyrinthine origin. Repeated convulsions in the chronic hypertensive patient are due to a previous cerebral softening or hæmorrhage or to unrelated brain disease. Most instances of "vasospasm" occur during the final thrombotic closure of vessels seriously narrowed by atherosclerosis but a full account of what is called vasospasm would take us too far afield at present.

Thus chronic hypertensive encephalopathy becomes a matter of repeated small strokes with or without dementia and no particular disadvantage attaches to the use of the term, if the underlying pathological basis (*état lacunaire* or small hæmorrhages) is kept clearly in mind. It must be remembered that cerebral embolism frequently complicates hypertension, the embolus usually arising from a diseased heart. Often entirely unrelated diseases such as berry aneurysm, tumour, abscess, meningitis and senile or presenile dementia must first be ruled out before the illness is ascribed to hypertension.

For this work laboratory facilities were provided in the Department of Pathology at The Montreal General Hospital through the initiative and kindness of Dr. Joseph Pritchard and Dr. Francis MacNaughton. Dean Lyman Duff was most generous in his support of the project. This paper is in part a report of the work carried out there in the past three years.

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AFTER EFFECTS OF WESTERN EQUINE ENCEPHALOMYELITIS INFECTION IN MAN*

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THE HISTORY of western equine encephalomyelitis (W.E.E.) in Saskatchewan does not reveal when the first human cases occurred in this province. Most of the early cases had been regarded as non-paralytic poliomyelitis but doubt arose when they occurred in increasing numbers and frequently in districts free from typical poliomyelitis. Attempts, therefore, were made to determine the true nature of the disease by the isolation of the causative agent.

In 1939 the virus of W.E.E. was recovered from two cases of the hitherto undiagnosed disease, while neutralization tests done on others proved that they too had suffered from the same condition. Since that time cases have been diagnosed each year by virus isolation or by means of neutralization tests against the equine virus. Some seasons the disease assumes epidemic proportions as it did in 1938, 1940, 1941 and again in 1947, while in other years isolated cases appear over widely scattered areas throughout the Province.

Although there has been no serious outbreak of W.E.E. during the past few years and the death rate is much lower than it was in early outbreaks, the disease is still important from a public health point of view but unfortunately its real seriousness is not yet fully appreciated. It is common knowledge that paralysis may follow poliomyelitis and unless proper treatment is instituted immediately permanent injury may be expected. Because of this, poliomyelitis is more generally feared than is W.E.E. and during outbreaks extreme efforts are made to prevent the spread of the disease.

It is not yet generally known that the after effects of W.E.E. may be even more serious than those resulting from poliomyelitis. It has been realized for some time by doctors and those engaged in the study of this disease that after apparent recovery there may develop symptoms indicative of injury to the central nervous system. That mental disorders may result from infection by the equine virus was first suspected from cases occurring in infants. In some of these

*From The Virus Laboratory, University of Saskatchewan, Saskatoon.

patients mental changes occurred almost immediately after the acute symptoms had subsided. One of the first early cases of this type was that of a child nine months old who had been quite normal prior to contracting the disease. Proof that we were dealing with W.E.E. was obtained by neutralization tests, a negative reaction being secured when the disease was first suspected and a positive after fourteen days. A year later there was no improvement in the mental condition of the child and when death occurred a short time afterwards the brain showed changes typical of western encephalitis.

Although these mental changes were observed in children quite early in the course of our study it was not until a survey was made to determine the fate of adults who had suffered from the disease that it was realized that many had been similarly affected. Mental impairment in adults due to infection with the equine virus in most cases develops graduallly, and perhaps not for a year or more after recovery does the patient exhibit symptoms sufficiently marked to demand medical aid. In the course of the survey the co-operation of the family doctor was sought and an attempt made to obtain follow-up reports on all cases confirmed in our laboratory over the twelve year period from 1940 to 1952. As was to be expected, many of the earlier patients could not be traced, but a surprisingly large number of those where a history was obtained had developed mental symptoms and later died. Sufficient information was secured to show that the after effects of W.E.E. are much more common and serious than was hitherto thought. Of 101 cases, the information obtained showed that 15 presented the symptoms listed in the table.

Information received from one doctor is as follows:

"I may say that during the epidemic we had some thirty odd cases with W.E.E. There were about six deaths. One child who recovered had epileptic seizures. About four of the elderly people all showed signs of fairly rapid senility after recovery following W.E.E. Another case was that of a little girl who developed facial paralysis at the time of the infection." (The outbreak this doctor referred to was that of 1941.)

Considering that known cases of W.E.E. have terminated in mental disturbances sufficiently severe to require confinement in mental hospitals, and that mild cases of the disease might have gone unnoticed or have been confused with more benign conditions, it appeared that there might be patients in mental hospitals as a result of W.E.E. in whom the primary cause had not been suspected. This possibility seemed further strengthened since as already mentioned, the secondary condition in adults may be so delayed that the association might not be realized. Having this in mind, arrangements were made to test patients in the two mental hospitals in the Province, one of which is located at Battleford in the north, and the other in the south at Weyburn.

Patient	*Year disease contracted*	*Doctor's remarks*
C.M.	1947	In the past two years this man has been confined off and on in the Weyburn Mental Hospital and has received shock treatment for schizophrenia.
M.K.	1942	Acts like early post Parkinsonian.
N.N.	1943	Post Parkinsonian definite and of such severe proportion that he was forced to resign three years ago as a parts man in an implement shop.
J.T.	1942	Had definite mental retardation for a year prior to death.
M.J.	1949	Had anxiety neurosis 1949—improved in 1950.
G.P.	1948	Mild personality change.
R.G. (child)	1941	Seen November 12, 1942—backward.
E.F. (child)	1942	Seen September 1949—epileptic seizures.
B.R. (child)	1941	Cerebral palsy.
L.S. (child)	1949	Cerebral agenesis—died.
H.P. (child)	1948	Cerebral palsy.
G.H. (child)	1948	Cerebral palsy.
D.B. (child)	1947	Mentally retarded.
Y.K. (child)	1947	Mental retardation.
W.S.	1947	This man was a farmer who worked hard and showed keen interest in district affairs prior to having had W.E.E. About two years ago he changed very markedly and ceased to be interested in his farm or people who had known him for years. At times his memory was very poor. Examined in September, 1951—was well nourished but not normal mentally. Died in October, 1951.

It should be stated that prior to the testing of patients at these hospitals, considerable work had been done to determine if many healthy individuals in the Province harboured neutralizing bodies for the equine virus. Results obtained from this survey showed that of 1,700 tests done one gave a slightly suspicious reaction, while all others were negative. These samples were collected in widely separated districts throughout the Province; some from areas where the disease had appeared epidemically; others came from areas where isolated cases had been diagnosed;

while others were secured from parts where W.E.E. had not been recognized. From these findings it appeared that a positive reaction would have significance and could be accepted as proof of infection with the equine virus.

Tests were first taken at the northern hospital at which time there were some 1,500 patients. As would be expected, ages covered an extensive range from about 20 to over 80 years. Some of the patients had been confined for thirty years or more while others had been recently admitted. At first it was planned to test only those showing symptoms which could have resulted from an encephalitis and where the duration of the illness was such that positive reactions to the equine virus could still be expected if such an infection had actually occurred. As the work progressed however, it was decided to test all the patients in the hospital and to take into consideration the length of time the patient had been confined, which would be a factor in determining whether or not neutralizing bodies could be expected in the blood in measurable amounts. It is true that the length of time neutralizing bodies remain in the blood is quite variable, and to some extent limits the scope of work of this nature. We find that it is not at all uncommon to secure a strong positive reaction six years or more after recovery from W.E.E. In one case where there were no ill effects following an attack of the disease a strong positive reaction was obtained twelve years after recovery. The neutralizing titre of the blood in this case has not changed markedly since the first time a test was made. This is perhaps an unusual case, at least in our experience it is the longest time over which neutralizing antibodies have persisted after known infection.

In view of these facts, and considering that W.E.E. was first recognized in Saskatchewan some fourteen years prior to the commencement of this study, it seemed reasonable to expect some positive reactions would be secured if the equine virus had played any part in the patient's mental condition. It has been noted during the course of this work that there is, in most cases, an inverse relationship between the duration of the mental illness and the neutralizing quality of the blood. Patients who have been ill for a number of years more often give a weak positive or partial reaction while the blood from more recent admissions is usually quite high in neutralizing qualities.

The work commenced at the Battleford Hospital in January, 1950 and carried on until April, 1951 and over 1,500 tests were made. Some patients had been in the institution since 1914, others being admitted each year from that time until the present. All those tested who had been admitted to the hospital between 1914 and 1932 proved negative while of those admitted in 1933, one gave a reaction which was suggestive of having been infected with the equine virus. Two patients hospitalized in 1942 gave incomplete results, as did one of the 1947 group together with two admitted in 1949. Twelve of the 1,500 or 0.08% gave very strong positive reactions proving definitely they had been infected with the virus of W.E.E. These patients were admitted as follows: one in 1937, one in 1942, one in 1946, two in 1947, one in 1949, two in 1950 and four among patients admitted between January 1 and March 31, 1951, when the general testing at the Battleford Hospital was completed. The high percentage of positive cases located in the early months of 1951 stimulated a further interest in the matter and since the authorities at the hospital were keenly interested in the results secured throughout, they suggested that the work be continued testing all new patients on arrival. This plan was agreed upon, and from April 1, 1951 until October 10, 1952, 619 examinations were made, fifteen or 2.42% of which proved positive while three or 0.48% were classified as incomplete or suspicious.

Turning our attention to the Weyburn Hospital we found conditions much the same as at Battleford. Many of the patients had been hospitalized for such a long period of time that positive reactions could not be expected even though they had been infected with the equine virus prior to admission. Further, many of the early patients had been confined to hospital before W.E.E. had been recognized in the Province and any cases which might have developed after admission would most certainly have been recognized. Considering these facts, and realizing that negative results were obtained at Battleford with patients who had been confined prior to 1933 it was decided to examine only those who had been admitted after W.E.E. had appeared in the Province. In this group there were 189 on which neutralization tests were carried out. Nine or 4.76% proved positive while one gave a suspicious reaction. The high percentage of positive cases located at Weyburn is not surprising, since,

as already stated, the patients considered were only those who had been hospitalized after W.E.E. became common in the Province. It should be also stated that the disease was first recognized in the Weyburn area and it has been the centre of outbreaks occurring in the Province since that time. At a later date a number of the early admitted patients were tested at the institution and, as at Battleford, only negative reactions were obtained. It will be noted that the majority of positive cases located at both institutions were among the more recent admissions. At Battleford, of 27 positives six were among those hospitalized between the years 1937 and 1947; while 19 were admitted during 1950, 1951 and up until October 1952. Of the nine positive cases at Weyburn three were admitted between 1942 and 1949 and six during 1950 and 1951.

It is difficult to interpret the significance of the high incidence of positives among the more recent admissions. There had been no outbreak of epidemic proportions among humans since 1947.

Two factors add to the difficulty. One, the long interval that may elapse between the primary infection and the onset of mental symptoms in adults. The other, the gradual character of the onset. The importance of this is drawn in the following table:

Patient	*Positive W.E.E.*	*Appearance of mental symptoms*	*Hospitalized*
J.S.	1948	1949	1949
J.M.	1944	1947	1949
W.	1941	1944	1945(died)
R.F.	1946	1948	1950

Because of this inconsistency of time elapsing between infection with the equine virus and the appearance of the mental symptoms, these positive cases cannot be associated with any particular outbreak of the disease. It is of interest to note, however, that of the 15 cases located at the Battleford hospital between 1950 and 1952 12 came from districts where the 1947 outbreak was quite severe. It is perhaps of further interest that prior to 1947 W.E.E. had never been reported from the municipalities in which these patients had resided. With regard to the nine positive cases located at Weyburn it is noted that all of these people came from districts where W.E.E. had appeared epidemically since the disease was first recognized in Saskatchewan. It would therefore be quite impossible to even hazard a guess as to when the primary infection occurred.

Not only has it been shown that mental derangement may follow infection with the virus of W.E.E. but according to Ayres and Feemster[1] the same condition may result after an encephalitis caused by the virus of eastern equine encephalomyelitis. These workers stated, "After the 1938 outbreak of eastern equine encephalomyelitis in southern Massachusetts, 34 persons were infected, 70% being under ten years of age; nine survived and six had permanent sequelæ after one year. One of the nine survivors cannot be located; two of the eight other survivors had died; four are hemiplegic, mentally deficient and emotionally unstable; one is mentally deficient, epileptic and hysterically inclined; one has a slight Friedreich's foot and is a habit problem; only one has made an apparently complete recovery."

Though there are many points still to be explained, these findings bear out the suggestion that not only do recognized cases of W.E.E. tend to undergo mental deterioration requiring detention in mental institutions but there are in our mental hospitals a considerable number of cases in which the mental breakdown can reasonably be attributed to a previous but undiagnosed infection with the equine virus.

SUMMARY

A disease considered as non-paralytic poliomyelitis appeared in Saskatchewan in 1935 becoming more prevalent each year until 1938 when it assumed epidemic proportions. Since many of the cases that year were in areas which were free from poliomyelitis it was decided to study the condition with a view to determining the causative agent.

During the summer of 1939 it was proved that the disease was in reality W.E.E., the virus being isolated that year from two typical cases.

As the study progressed it was observed that young infants after recovering from the acute condition, in many instances failed to develop mentally while older children showed an early mental deterioration.

It was later found that some adults became mentally ill after having suffered from the virus disease, the symptoms appearing a year or more after the primary infection.

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These findings suggested that there might be patients in our mental hospitals as a result of infection with the equine virus but the primary cause not suspected.

Arrangements were made to test patients at the two mental hospitals in the Province with the result that at one institution 27 positive and six suspicious cases were located. At the other hospital nine positive reactions were obtained while one gave an incomplete or suspicious result.

The co-operation of Dr. U. Gareau of Regina who first drew attention to mental changes in children is appreciated.

We are indebted to Dr. M. DeMay, assistant superintendent of the Saskatchewan Hospital at Battleford, for his interest and assistance when testing patients at that institution. Note: a grant from the Dominion-Provincial Public Health Research Fund made this work possible.

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PRIMARY PERITONITIS

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IDIOPATHIC cryptogenic or primary peritonitis, long recognized as a clinical and pathological entity is only infrequently considered in surgical journals. Maingot and others remind us that "primary" is the term given to those cases of peritonitis in which no obvious intra-abdominal cause is found, and which are in fact secondary in nature, as the infecting organisms are brought to the peritoneum from some distant focus by the blood or lymph channels or even the female genital tract.

Before the advent of chemotherapy the prognosis was bad, the fatality rate averaging about 76% (Budde) (Barrington-Ward). With the antibiotics available today, plus early diagnosis and proper treatment, deaths should be practically nil from this affliction.

Here follows a case report from the Southend General Hospital, Essex, England, in 1952.

A 41 year old multipara was brought by ambulance to hospital at 4.35 p.m. complaining of severe abdominal pain. I visited her about 12 minutes after her admission to the ward.

The family history was unremarkable; patient's past illnesses were limited to an appendicectomy several years before with no sequelæ.

The present sickness had begun that morning; while on a holiday at the sea front she was seized with pain in her lower abdomen which had spread and become excruciatingly constant since then. There was no vomiting, no bowel or bladder disturbances.

Her last normal period was completed 2 days before; she didn't think pregnancy was at all probable. She had been perfectly well the previous day.

She lay with legs flexed, moaning and occasionally writhing about. Her expression was anxious, the face flushed, forehead covered with cold perspiration, tongue moist but slightly furred. The pulse was 83, respirations 21, mostly thoracic but the diaphragm had some movement. B.P. 135/80.

The important findings were in the abdomen. There was no distension nor retraction in its lower half. It was exquisitely tender everywhere. There seemed to be more rigidity just above the pubes but the entire musculature was practically boardlike. Rebound tenderness was worse in the lower half of the abdomen. Percussion revealed no abnormal resonance or tympanitic sounds and auscultation suggested no outstanding diminution of intestinal sounds.

Digital vaginal examination produced nothing conclusive—only the same generalized pain on bimanual. Dark red blood on the glove, made me suspect the possibility of ectopic, albeit the patient's history was almost a denial of it. Rectal examination otherwise was negative.

Red cell count was within normal limits; urinalysis negative (catheter specimen) and white cells up to 11,700.

The pain did not alter in degree when the patient sat up nor was there any periumbilical blueness of the skin as sometimes occurs in acute pancreatitis; the patient had no history of digestive upsets, making stomach or gall bladder perforation a bit remote. A pertinent point in the recent history was her eating of shrimps, mussels, cockles and jellied eels the day previous to admission, but the pulse was too fast and the onset too sudden for typhoid.

The gynæcology consultant reported: (1) that the cervix did not appear pregnant; (2) that the reproductive organs were probably quite normal; (3) that he could not support a tentative diagnosis of ectopic gestation.

In view of the doubt as to the nature and origin of the peritonitis, laparotomy was performed through a right paramedian incision. The naked eye findings of the extraperitoneal tissues were negligible. On opening the peritoneum, however, there exuded about four ounces of somewhat watery but slightly purulent yellowish gray odourless fluid, which unfortunately immediately found its way into the waiting suction apparatus and was never recovered for bacteriological study. No more fluid was found either free or isolated in the abdominal cavity.

The subsequent exploration revealed the absence of the appendix and the following structures were normal: large and small bowel, liver and biliary system, stomach and duodenum, kidneys, bladder and ureters, uterus and adnexa; the posterior abdominal wall was free of inflammation, pus pockets and neoplasms. In short we had a case of acute diffusing primary peritonitis. The peritoneum itself at the site of entry had lost its natural sheen and a surrounding area of about 20 centimetres was injected but the frosted appearance had not yet become striking.

Closure was effected in layers without drainage. (In my humble opinion drainage is overdone today; generally speaking there has not been a diminution of this practice commensurate with our advances in anti-infection measures.)