A Report of Two Outbreaks of Equine Encephalomyelitis in Saskatchewan

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DURING the early part of August 1935, reports were received from the northern states of the Union and from the province of Manitoba that a great many horses were suffering from a disease believed to be encephalomyelitis. Shortly after the reported outbreak in Manitoba, isolated cases of the same disease were recognized in south-eastern Saskatchewan, and in the course of a week to ten days the outbreak had assumed quite serious proportions.

From the south-east the disease spread rapidly westwards, north, and towards the central sections, until in a short space of time cases could be observed one hundred to one hundred and fifty miles from the original outbreak.

A journey through the districts where the outbreak was most severe revealed the fact that, although the disease had visited nearly every farm and attacked animals of all ages, seldom did it affect more than one or two individuals on any one holding.

To go into a detailed description of the symptoms presented by animals suffering from the disease would be quite unnecessary. It may be stated, however, that they were identical with those so often described as characteristic of the once obscure conditions spoken of as forage poisoning, cerebro-spinal meningitis, corn-stalk disease, blind staggers, and similar to those of botulism.

Although the diagnosis arrived at in the adjoining States and Province was evidently quite acceptable in Saskatchewan, it was immediately realized that, owing to the magnitude of the outbreak, a definite diagnosis was imperative, and that such could not be made until the presence or absence of the virus of encephalomyelitis was demonstrated.

The fact that the majority of affected horses in Southern Saskatchewan were running on pasture where grazing conditions were identical, although quite unusual, brought to mind the work of Graham (1) on so called corn-stalk disease. Owing to the prolonged drought, wheat crops were considered a failure; and, when a few inches tall, were made use of to pasture live stock. Just prior to the disease outbreak, rains had fallen, resulting in rapid growth of some grain and deterioration of the older plants. Although these conditions were at first thought by some to have a bearing on the disease outbreak, it soon became apparent that another cause must be found. From the drought areas, the disease spread into districts where rainfall had been normal and feed plentiful; and, although there the epidemic was not so severe as in the drier sections, sufficient cases developed to show quite clearly that the feed situation had no bearing on the presence of the disease.

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The epidemic increased in intensity until a peak was reached about the middle of September. From that time fewer new cases were reported, and the spread of the disease was much less rapid. Towards the end of September, several frosts occurred, and almost immediately the outbreak was over.

Post-Mortem Examinations

Post mortems held on a large number of animals showed that pathological changes were confined to the brain. Gross examination of the brains of animals which had died early in the course of the disease often revealed a slight congestion which was usually confined to one cerebral hemisphere. In these cases, the cerebellum and upper extremity of the cord could not be considered as abnormal.

Where the duration of the illness had been prolonged, more marked tissue changes were observed. The surface cerebral congestion was less constantly present, but as a rule the cut surface showed changes which, in some instances, were so extensive as to involve the greater part of the hemisphere. The areas referred to were decidedly soft, yellowish in colour, and the smaller ones often completely surrounded by a hyperaemic zone which was less distinct or totally absent where a large part of the brain tissue was involved.

The degenerative changes referred to, in some instances, invaded both hemispheres, but more often only one was affected.

The microscopic examination of the cerebrum showed areas of congestion, oedema, and necrosis; but the general picture was one of congestion and haemorrhage.

Experimental Transmission

When the transmission of the disease was first attempted, blood was secured from field cases during the febrile stage, and inoculated subcutaneously, intraperitoneally, and intramuscularly into rabbits and guinea-pigs. One healthy horse was also used, blood being introduced subcutaneously and intravenously. Although a number of such attempts were made to transfer the disease, negative results were obtained in every case.

It was now decided to make use of brain material for inoculating purposes since it was appreciated that the virus of encephalomyelitis was likely to be much more constantly present in this organ than in the peripheral circulation.

Brains were removed from a number of animals which had just died, and from some which had been destroyed in the later stages of the disease, the surface washed in two per cent phenol, and the organ stored at ice box temperature in equal parts glycerine and Ringer's solution. Sections of the brain tissue were ground in normal saline, a $\frac{1}{20}$ suspension prepared, and varying amounts inoculated subcutaneously and intraperitoneally into experimental animals. Although several brains were used and a series of guinea-pigs inoculated with each, it was found impossible to reproduce the disease.

Suggestions were received in correspondence from Dr. Karl F. Meyer, San Francisco, as to how best to secure suitable brain material, the part to

be used for preparing the emulsion, and the method of inoculating experimental animals.

Horses in the early stages of the disease were destroyed, the brains removed and preserved in the glycerine and Ringer's solution.

Sections of the medulla were used in preparing the $\frac{1}{20}$ emulsion, 0.2 cc. of which was introduced intracerebrally into guinea-pigs weighing from 250 to 350 grams. The fifth attempt with this technique produced in a healthy guinea-pig elevation of temperature on the third day, when the animal was destroyed and the brain removed, prepared in the usual manner, and introduced into four guinea-pigs by the intra-cerebral route. Only one of the inoculated animals became infected; but, when this brain was used for further inoculations, the virus was evidently firmly established, as from that time on no difficulty was experienced in reproducing the disease. It should be here stated that all brains removed from guinea-pigs were tested for sterility before making use of them for further inoculating purposes.

During the early stages of the experimental work, a number of animals died from shock almost immediately after inoculation, but with improved technique such mortality was reduced to practically nothing.

The early technique consisted of giving a general anaesthetic after the skin over the forehead had been shaved and scrubbed with 70 per cent alcohol. An incision was made about the centre of the forehead, running parallel to the median line, the skin retracted, and with a fine drill and small brace an opening made through the bone exposing the brain tissue. With a half-inch 26-gauge needle, the inoculum was introduced directly into the cerebrum, the skin incision brought together with silk sutures, and adhesive tape applied over the wound.

It was later found that infiltration of the skin of the forehead with novocain was much more satisfactory than the use of a general anaesthetic, and it was also found that the drill was unnecessary and that an opening could be made with an 18-gauge needle when rotated back and forth between the thumb and forefingers. With this technique, one can determine when the bone has been penetrated much better than when a drill and brace are used. With young guinea pigs no difficulty is experienced in passing the fine gauge needle used for inoculating directly into the brain tissue when the needle is introduced at the point of the bone suture, but this technique often results in the death of the animal, probably due to intracranial pressure which is partly relieved by the larger opening.

After the virus had been passed through a series of three guinea-pigs, the latest brain was prepared and 9 cc. of a $\frac{1}{20}$ suspension introduced intracranially into a healthy horse. The animal showed marked symptoms of the disease on the fourth day, was destroyed on the fifth day after inoculation, and the brain removed for further guinea-pig inoculations. At this time a sheep was also inoculated with virulent brain material, but the animal remained well.

From the results obtained, it appeared we were dealing with a virus disease, and it was felt reasonably safe to conclude, equine encephalomyelitis.

It was decided to make an attempt to determine the type of virus, and with this end in view a series of guinea-pigs were inoculated with Eastern and Western Commercial Anti Equine Encephalomyelitis Serum. Although a large number of animals were so treated, and in some cases with serum doses out of all proportion to the size of the animal, it was found impossible to protect against the Saskatchewan virus. Brain material was forwarded to Dr. Karl F. Meyer, which he tested against antiserum prepared at his laboratory and found to be neutralized with the Western type. He also found that the Saskatchewan virus was particularly virulent and much more difficult to neutralize than some of the strains found in the United States.

After it had been definitely determined that we were dealing with the Western type, further attempts were made to protect experimental animals with commercial antiserum, but the results obtained were completely inconclusive. At this time, commercial vaccine was also made use of, but again it could not be stated that the vaccine was of any value in protecting experimental animals. On the contrary, we found that one commercial vaccine, when introduced subcutaneously into a group of four guinea-pigs, produced symptoms of encephalomyelitis in three days; and, when the brains from these animals were used for subsequent inoculations, the presence of a quite virulent virus was demonstrated.

The different methods of immunizing guinea-pigs, as described by Howitt (2), Records and Vawter (3), have been tried with the Saskatchewan virus, and the results obtained will be reported on at a later date.

The Outbreak of 1937

Although a number of cases of encephalomyelitis appeared throughout the Province during the late summer and fall of 1936, nothing approaching an epidemic occurred. On July 20, 1937, the presence of an unusual disease was reported from the central sections of Saskatchewan, and upon investigation it was found that a number of horses had been lost after a few days' illness. The only surviving animal, a yearling colt, which had been sick for five days, was found in a partially paralyzed condition and unable to stand. The symptoms presented by the colt strongly suggested encephalomyelitis, but no confirmation was obtained.

On July 25, several cases suggesting encephalomyelitis were observed in the same district, and towards the end of July became quite prevalent within this area. About the time of this local outbreak, with astonishing suddenness the same disease appeared in epizootic form in the extreme south-eastern part of the Province, and in the same district in which the 1935 outbreak of encephalomyelitis originated. From this focus the disease again radiated out towards the north, west, and central parts of the Province, at first advancing with a solid front, but later isolated outbreaks occurred perhaps fifty miles from the advance line of the original outbreak.

By the middle of August, cases were reported from all sections of the Province except the extreme north-west where but few animals were affected. The epidemic was perhaps at its height during the first week in

September but from then on, although the spread continued, the disease appeared on fewer farms and affected fewer animals in each stud.

With the abatement of the outbreak, there was a marked decrease in the mortality rate, few animals succumbing even in cases where the early symptoms were pronounced and proper veterinary care not available.

That we were again dealing with encephalomyelitis was generally accepted, but owing to certain differences between the two epidemics, it was thought advisable to require further experimental proof.

Unlike the 1935 outbreak, the disease was more sudden in onset and more acute in nature, it being not at all uncommon for animals to become stricken while at work, succumbing in from twenty-four to forty-eight hours after the first signs were noted. Another point of difference was that in the former epidemic, seldom did more than one or two animals become infected on any one farm, while during the early period of the 1937 outbreak, in some instances, as high as 75 per cent of the horses on a farm suffered from the disease.

Owing to these differences, and bearing in mind the report of Marsh (4) on an outbreak among horses, clinically indistinguishable from encephalomyelitis, but lacking any demonstrable virus, it was considered necessary once more to prove the presence or absence of the virus.

An animal which had been sick but twenty-four hours was secured, and the brain removed in the usual manner. Fresh specimens of the medulla were emulsified, and a series of guinea-pigs inoculated intracerebrally. The first attempt failed, but material from the same brain reproduced the disease upon subsequent inoculations. Typical temperature curves were secured from the experimental animals, the symptoms of encephalomyelitis developed with paralysis of the hind quarters.

The new virus proved to be particularly virulent for guinea-pigs, in some instances, death occurring seventy-two hours after inoculation. Brain emulsion in dilutions of $\frac{1}{80}$, passed through a Berkefeld filter of N porosity, constantly produced the disease in guinea-pigs.

That equine encephalomyelitis has been common in the Province for a great many years, there is no doubt, although it was first definitely recognized as such in 1935. The writer has been called upon time and again to investigate local outbreaks, described as meningitis, forage poisoning, and in some instances, botulism. These outbreaks have been confined to certain areas in the Province; and, interesting to state, parts which have not been visited to any extent by the outbreaks under discussion. The small outbreaks of the past have occurred during the late summer and early fall, and there is now little doubt that in reality the disease was equine encephalomyelitis. This conclusion has been drawn in spite of the fact that it is appreciated that, whenever a new disease is recognized, there is always a great tendency to make use of it unjustifiably for the diagnosis of conditions which have given difficulty in the past.

It is true that in several instances outbreaks of botulism have been recognized from clinical symptoms and the organism isolated from spoiled feed, but very often this diagnosis has been used where the animals have

been running at pasture and spoiled feed was not available. It is true that in most cases botulism and equine encephalomyelitis may be differentiated by clinical symptoms, but it is also true that, in the acute forms of these diseases, the differential diagnosis becomes extremely difficult.

Treatment

With regard to the treatment of encephalomyelitis, it should be stated that during the outbreak of 1935 antiserum was used extensively, and considered by many practitioners as being a specific if used early in the course of the disease.

At the beginning of the 1937 epidemic, it was mentioned that the disease was particularly virulent and that the majority of affected horses succumbed. During that period anti-encephalomyelitis serum was used freely in many districts where the disease appeared, but to little avail, whereas later in the season after the outbreak had reached its peak, and when antiserum was not available the death rate fell very appreciably. The question, of course, arises: Would serum have further reduced the mortality? To appraise the value of treatment under field conditions is difficult, often impossible, but it can be stated that many practitioners secured as satisfactory results without antiserum as did those who relied upon it entirely in the control of the disease. It should be noted, however, that in the isolated outbreaks previously referred to there was a marked difference in mortality even when treatment was identical. Evidently variations in virulence of virus or resistance of host have much more to do with the death rate than has the form of treatment adopted.

Seasonal and Geographic Distribution

Equine encephalomyelitis is definitely a seasonal disease, appearing during the late summer and fall months. Outbreaks commence suddenly, reach a peak, taper off gradually until the weather becomes frosty, when with decided abruptness the disease disappears.

The incidence of encephalomyelitis is evidently not governed by the topography of the country, since the outbreaks under discussion travelled from the dry plains of the south, through the rolling park country, into the timbered lands of the north where lakes and swamps are numerous.

The 1935 outbreak commenced in a territory which had been without rain for months, and spread and flourished equally well in the north where rainfall was above normal.

Natural Transmission

Although it has been shown by Records and Vawter (5) that encephalomyelitis may be transmitted by nasal washings from infected horses, it would appear very doubtful if the disease is commonly transferred in the field by direct contact.

From observations during the Saskatchewan outbreak, little or no attempt was made to isolate the sick; in fact, a common practice was to allow infected animals the freedom of the farm yard and permit them the use of the common drinking trough. In spite of such methods, further cases sel-

dom developed on these farms. It is true that during the 1937 outbreak on some farms 75 per cent of the horses became infected, but in the majority of such instances the animals contracted the disease about the same time, and probably from the same source rather than from contact with one another. The appearance of the disease about the same time on farms and in districts many miles apart where even indirect contact was out of the question would also suggest that contact is not of major importance. Furthermore on certain farms in districts where encephalomyelitis was prevalent and where direct contact with infected animals was established, cases did not develop.

Since it had been shown by Kelser (6), Madsen, Knowlton and Rowe (7) that encephalomyelitis could be transmitted experimentally by means of mosquitoes, this manner of transfer was continually kept in mind.

As has been heretofore stated, the southern section of Saskatchewan was particularly dry during the season of 1935, while the entire area through which the disease traversed in 1937 had been without rain for most of the summer. Owing to the prolonged drouths, only in the early part of the season were mosquitoes at all present, and even then not numerous. For many weeks prior to and during the period when the disease was prevalent, mosquitoes were altogether absent and could, therefore, be definitely eliminated as a factor in transmission.

Throughout the course of the epidemics, the only biting fly recognized was *Stomoxys calcitrans,* and this fly was particularly prevalent during the late summer and fall of 1937. Animals running at pasture were kept in a state of unrest, and in some sections the farmers had to resort to the use of sprays to protect stabled live stock.

In previous years these flies were never so numerous in Saskatechewan as to be annoying to humans; in fact, seldom did one ever observe their presence. During the month of August 1937, the stable fly became so prevalent in certain sections that one could not remain outdoors without being bitten.

The fly in question disappeared after a few night frosts late in September, and almost at the same time did the equine disease.

Even although one may be justified in suspecting *Stomoxys calcitrans* as a possible means of transmission, the same objection may be taken to this theory as was taken to the method of spread by direct contact. On many ranches, where large bands of horses were running together with no fly protection whatever, but few cases would develop. The disease might miss adjoining ranches entirely to reappear again perhaps fifty miles distant.

On many farms in Southern Saskatchewan about the middle of August conditions were quite comparable to those which might be planned experimentally with regard to transmission by means of these biting flies. Owing to the lack of crop in the area, no farm work was being done with the result that all horses were running at large both day and night. Time and again infected horses could be seen running with the healthy, and during the day all animals would be literally covered with the fly in question. In spite

of this fact, as already mentioned, seldom did new cases develop after the disease first visited the premises.

Even when it is appreciated, as has been shown by Howitt (8), Records and Vawter (9), that only in the early stages of the disease is the virus present in the peripheral blood circulation, it is difficult to understand why the disease is not more often transferred.

Summary

Equine encephalomyelitis was first recognized in Saskatchewan as such in 1935 when the disease appeared in epidemic form.

A second outbreak occurred in 1937, which was more extensive than the first epidemic and responsible for many more deaths.

The outbreaks referred to occurred during the late summer and fall months and covered much of the same territory.

The virus was found to be present in the brain of field cases, which were destroyed early in the course of the disease, but could not be demonstrated in the peripheral blood circulation even when samples were secured immediately after the onset.

Experimental transmission was accomplished by inoculating guinea-pigs intracerebrally with brain emulsions prepared in normal saline solution.

The virus was found to pass through a Berkefeld filter of N porosity.

Although equine encephalomyelitis was first recognized in Saskatchewan in 1935, it has no doubt been present for many years, and described as forage poisoning, cerebro-spinal meningitis, blind staggers, or sleeping sickness.

The writer is grateful to Dr. Karl F. Meyer, San Francisco, and Dr. E. Records, Reno, for valuable suggestions which were freely given.

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