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VIRUS MENINGO-ENCEPHALITIS IN AUSTRIA

1. Epidemiological Features

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SYNOPSIS

This paper reports on an epidemiological study of 304 cases of virus meningo-encephalitis hospitalized at the Neurological Clinic at Graz during the 1953 epidemic in Styria, Austria. The epidemic showed a distinct seasonal periodicity, with an increase in the number of new cases during the period June-September; the monthly percentage of paralytic and encephalitic cases was, however, lowest in July and August. While no epidemic centre could be distinguished, 78% of all patients came from rural, usually wooded, areas. The maximum morbidity occurred between the ages of 11 and 30 years. The severity of the disease increased with age, save for the group 1-10 years old.

A history of insect bites was noted in a large proportion of patients, one-third reporting tick bites, and many others, mosquito bites. The author finds both the period of incubation of the illness and the chain of infection to be very variable; there appeared to be cases caused by family contact, possibly through droplet- or dust-borne infection, and a possibility of infection through foodstuffs.

But for fatigue, concomitant illness or injury, and other precipitating factors, some cases might have been abortive; such factors also appear to determine the severity of the illness.

Assessment of the epidemiology of diseases of the central nervous system is fraught with considerable difficulty, since the etiology of many of the virus meningo-encephalitides observed with increasing frequency during the last few years remains unexplained. Consequently, the possibility of systematically classifying and comparing the various epidemics which continually flare up here and there in Central Europe is somewhat limited.

Although sporadic cases have been observed almost everywhere, distinct endemic regions have emerged in which, in many years, the number of cases has increased to epidemic proportions. The region surrounding Wiener Neustadt, south-west of Vienna, has been known as a permanent focus of meningo-encephalitis for more than 20 years. As long ago as

1932, Schneider [25] reported cases there and spoke of an epidemic of acute serous meningitis. In the neighbouring area of Baden, near Vienna, similar cases have been increasingly frequently observed during the last 10 years, and Drexler [2] has expressed the view that they probably result from virus forms of meningo-encephalitis, since attempts at culture have always proved unsuccessful and tests for the leptospiroses have been negative. Similar cases were described in Carinthia, the southern part of Austria, bordering on Yugoslavia, by Lasch & Moritz.[18] The virus meningo-encephalitides have also occurred with increasing frequency in Styria, being particularly prevalent in the eastern part of that province, where there was also a high poliomyelitis morbidity during the great epidemic of that disease in Austria in 1947.[10, 11] Epidemics of virus meningo-encephalitis have been observed, following the same course or a course similar to that in Austria, in other Central European countries. Kral[14] has described one such epidemic which affected about 1,000 persons in the Theresienstadt concentration camp in Czechoslovakia in the winter of 1943-4. A wave of encephalitis was observed in the Palatinate in 1947-9,[1] and a similar cluster of meningo-encephalomyelitis cases occurred in 1948-50 in the Aachen area.[32] The causative agent could not be isolated from any cases in these epidemics, and it should not be assumed that they were all caused by the same virus. However, Lépine [19] is of the opinion that the local epidemic in the Palatinate must have been a disease belonging to the Eurasian subgroup of the arthropod-borne encephalitides, since mosquito bites were repeatedly mentioned in the case-histories.

In 1948, local epidemics of meningo-encephalitis developed in Bohemia and Moravia and were carefully studied by Hloucal [7,8] and Krejci,[15] respectively. Sporadic cases of meningo-encephalitis were repeatedly recorded in the same regions of Czechoslovakia in the following years,[9] and Gallia, Rampas & Hollender [5] were able to isolate a virus from the blood of patients in the viraemia stage. In view of the similarity to Russian spring-summer encephalitis (RSSE), it was believed that the virus was transmitted by a species of tick,[16] as is the case with RSSE; [29] in fact, Rampas & Gallia,[22] and Krejci [17] independently of these two, were able to isolate a virus from the tick *Ixodes ricinus*. Edward [3] was able to show by serological tests that the virus of the so-called Czech tick encephalitis belongs to the group of arthropod-borne encephalitides.

This Czech tick encephalitis is of particular interest since it afforded the first proof that a virus of the arthropod-borne encephalitis group had caused an epidemic in Central Europe. The only virus of this group previously detected in a few sporadic human cases in Central Europe had been that of louping-ill.[33]

An epidemic of meningo-encephalitis developed in Styria in 1953, and the causative agent has been shown by Verlinde and his co-workers to be a virus of the arthropod-borne encephalitis group (see page 565).

The clinical picture of this disease is given by Grinschgl on page 535. It is, of course, impossible to decide in retrospect whether the epidemics of meningo-encephalitis which occurred in previous years in Austria and other Central European countries were also caused, at least partly, by the same virus. However, it is certain that the epidemic in Slovenia which occurred at the same time as the Styrian epidemic is of similar origin.

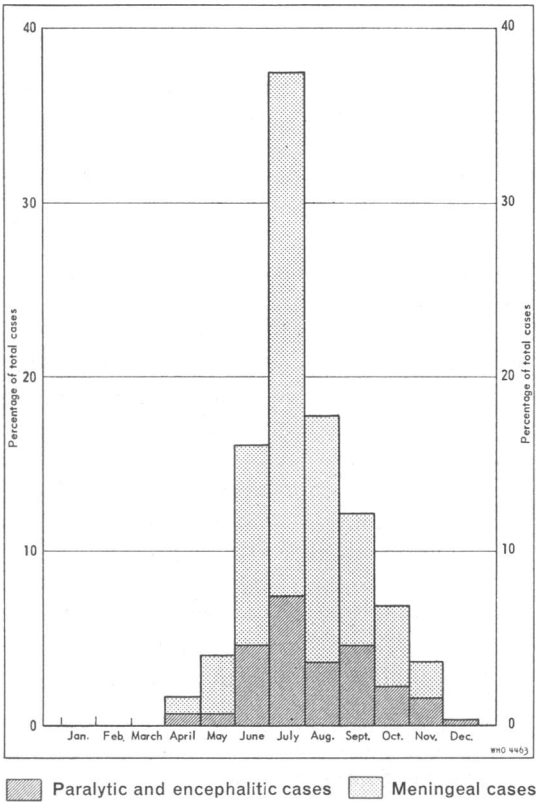
The following survey of the epidemiological characteristics of the Styrian epidemic refers only to the 304 cases admitted to the Neurological Clinic at Graz. However, hundreds of similar cases were admitted at the same time to the smaller provincial hospitals; we were unable to study these cases, since no virological studies were made on them and since they were often diagnosed as poliomyelitis. Moreover, most of the purely meningeal forms failed to come to the notice of the health authorities, since the Austrian law on epidemic diseases provides for the compulsory notification of " communicable infantile paralysis " (poliomyelitis) and " communicable encephalitis " (epidemic encephalitis) only, but not of other virus meningitides and encephalitides. This hiatus in the legislation, and the practice followed in provincial hospitals whereby paralytic cases are notified as poliomyelitis before the etiology has been clearly determined, explain why the official statistics of infectious diseases were useless for our purpose.

Three sporadic cases which occurred outside the epidemic period and were admitted to the clinic in January and February 1953 have been included in the tables and figures showing seasonal incidence, but they will not be considered in detail since presence of the virus was not confirmed and it cannot be definitely stated that they were caused by the same agent.

Seasonal Distribution of Epidemic Cases

The epidemic under consideration showed a distinct seasonal periodicity, with an increase in the summer months from June to September similar to that found with other forms of arthropod-borne meningo-encephalitides (see fig. 1). Three sporadic cases, one of them fatal, were admitted to the Neurological Clinic at Graz in the first two months of 1953. The first five epidemic cases were admitted in April; one of these, with a clinical picture of ascending Landry paralysis, died of bulbar paralysis. In May there were 12 new cases, one of which was fatal. Up to this point, there was no notable increase over the number of virus meningo-encephalitis cases usually occurring in the early summer. In June, however, 49 new cases were admitted, one of them fatal. The peak of the epidemic was reached in July with 114 new cases, four of which died. In August, the number of new cases fell to 54, three of which were fatal. In September, October, and November, the number of new cases fell to 37, 21, and 11, respectively, two being fatal in September and one in November. One

FIG. 1. MONTHLY INCIDENCE OF VIRUS MENINGO-ENCEPHALITIS IN STYRIA, 1953, EXPRESSED AS PERCENTAGE OF 304 CASES ADMITTED TO NEUROLOGICAL CLINIC, GRAZ



fatal case occurred in December. The same general picture emerges from the weekly totals, the maximum number of new cases occurring in the 29th and 30th weeks.

There was no particular variation within each month in the distribution of cases by sex (see table I).

The distribution of paralytic and encephalitic and of meningeal forms in the various months is of particular interest. The percentage of paralytic and encephalitic cases was smallest (20% of the total monthly cases) in July and August—at the height of the epidemic. In June and September —the two months with the next highest numbers of cases—the severe forms amounted to 29% and 38%, respectively. In the autumn months of October and November, there was, at the same time as a fall in the number of new cases, an increase (up to 46%) in the proportion of paralytic and encephalitic forms; a similar situation occurred at the beginning of the epidemic.

TABLE I. NUMBER OF VIRUS MENINGO-ENCEPHALITIS CASES HOSPITALIZED MONTHLY AT NEUROLOGICAL CLINIC, GRAZ, 1953

Month	Male cases			Female cases			Total male and female cases*
	paralytic and encephalitic*	meningeal*	total	paralytic and encephalitic*	meningeal*	total	
January **	2 (1)	0	2	0	0	0	2 (1)
February **	0	0	0	0	1	1	1
March	0	0	0	0	0	0	0
April	2 (1)	1	3	0	2	2	5 (1)
May	1 (1)	6	7	1	4	5	12 (1)
June	8	22	30	6 (1)	13	19	49 (1)
July	14 (2)	52 (1)	66	9	39 (1)	48	114 (4)
August	8 (3)	19	27	3	24	27	54 (3)
September	6	12	18	8 (2)	11	19	37 (2)
October	6	11	17	1	3	4	21
November	5 (1)	5	10	0	1	1	11 (1)
December	1 (1)	0	1	0	0	0	1 (1)

* The figures in parentheses indicate the number of fatal cases.
** The cases occurring in January and February are considered sporadic.

A study of the figures for poliomyelitis epidemics shows that these usually have a seasonal peak in Central Europe in September; in southern countries, however, this may occur earlier in the summer.[4] Czech tick encephalitis in 1948, like the Styrian meningo-encephalitis epidemic of 1953, showed a peak in July. RSSE reaches its peak somewhat earlier, probably because of different climatic conditions.

Geographical Distribution of Cases

The cases shown in fig. 2 refer solely, as has already been stated, to patients admitted to the Neurological Clinic at Graz; they therefore give a correct picture of the morbidity only for the areas of Styria close to Graz, that is, the immediate region served by the clinic. No distinct epidemic centre can be seen; the 17 cases admitted up to the end of May 1953 occurred apparently at random in the immediate surroundings of Graz, in a few more distant districts, and in a certain area of eastern Styria where clinically similar cases had repeatedly occurred in previous years. Since this latter area had been particularly affected by the 1947 poliomyelitis epidemic, there was a tendency to classify endemic cases observed there from 1948 to 1952 under the heading of poliomyelitis in the aparalytic and

FIG. 2. DISTRIBUTION OF 304 CASES OF VIRUS MENINGO-ENCEPHALITIS IN STYRIA, AUSTRIA, 1953

Each case is represented by a dot.

paralytic forms. However, in view of the discovery of the meningo-encephalitis virus in 1953 that classification must be considered doubtful.

At the end of June 1953, when a total of 66 cases had been admitted, there was a distinct concentration of cases in the same eastern districts of Styria, but new cases also developed in the immediate neighbourhood of Graz and in the city itself. The distribution of the 180 cases admitted up to the end of July shows a further concentration in those same districts and a merging of the epidemic areas in eastern Styria and in the neighbourhood of Graz. Occasional isolated groups of cases occurred to the west of Graz. By the end of August, 234 patients had been admitted, with a distinct concentration within the city of Graz and a small increase in southern Styria towards the Yugoslav frontier. The admission of 37 new cases in September did not change the geographical picture.

Approximately 75% of all the cases occurred in the area extending from eastern Styria into the city of Graz. While the map shows the heaviest concentration of cases near that city, which has a population of 280,000, the morbidity rate there is, in fact, distinctly lower than in the neighbouring and appreciably less thickly populated rural areas, whence most of the cases came. These rural areas were usually lower mountain slopes or thickly wooded hill regions. Frequently, the patients even came from particularly

remote and isolated areas, far from population centres. A similar tendency to occur in wooded areas is reported of RSSE. Thus, Smorodintsev [30] states that cases of the Far Eastern form of RSSE occur largely in wood-cutters' camps; its Western variant caused epidemics in 1944 in the wooded regions of Byelorussia [26, 28] and later in the Beloveja National Park.[6] The Czech tick encephalitis epidemic of 1948 also mainly affected the rural areas of southern Bohemia and Moravia. In the Slovenian epidemic, which occurred at the same time as the Styrian, forest workers were those predominantly affected (see page 491). A similar clinical picture was also observed in the summer of 1953 in other provinces of Austria—namely, Upper Austria, Lower Austria, Burgenland, and Carinthia.

Age Distribution

It is interesting to note the relatively small degree to which the lower age-groups were affected. Only 16 of our patients were between 1 and 10 years old, about half of those being of pre-school age and half of school age. These children represent little more than 5% of the total cases, and no deaths occurred among them. The maximum morbidity occurred between the ages of 11 and 30 years, with 73 cases in the 11-20 year age-group and 71 in the 21-30 year group; nearly 48% of the total cases thus occurred at these ages. As will be seen from table II the number of cases declined progressively in the subsequent 10-year groups.

TABLE II. AGE DISTRIBUTION OF 304 VIRUS MENINGO-ENCEPHALITIS CASES

Age-group (years)	Male cases			Female cases			Total cases		Total paralytic and encephalitic cases	
	paralytic and encephalitic	meningeal	total	paralytic and encephalitic	meningeal	total	number	%	number	%*
1-10	4	8	12	2	2	4	16	5	6	38
11-20	1	37	38	6	29	35	73	24	7	10
21-30	14	33	47	6	18	24	71	23	20	28
31-40	8	26	34	6	19	25	59	19	14	24
41-50	11	11	22	2	18	20	42	14	13	31
51-60	8	9	17	4	10	14	31	10	12	39
61-70	2	3	5	2	1	3	8	3	4	—
71-80	3	1	4	0	0	0	4	1	3	—

* Where the denominator is less than 10 the percentage has not been calculated.

Classifying the cases by age and sex, we find, for men, a gradual increase of cases reaching a peak in the third decade of life and gradually falling off in the higher age-groups. For women, the peak occurs in the second decade, the three subsequent 10-years periods showing approximately equal though smaller figures, followed by a distinct drop after the age of 51 years.

The percentage distribution of severe and mild forms within the various age-groups is worthy of notice. Thus, with children aged 1-10 years, severe paralytic forms are found in about 38% of cases. In the 11-20 year age-group, on the other hand, the proportion of paralytic and encephalitic cases is very small (less than 10%). In the third, fourth, and fifth decades of life the percentage of severe forms is approximately equal (25%-30%).

TABLE III. AVERAGE AGE OF CLINICAL CASES

Course of disease	Number of cases			Average age (years)		
	male	female	total	male	female	total
Meningeal	128	97	225	28.4	30.8	29.4
Paralytic	42	15	57	39.7	28.9	36.9
Encephalitic	9	13	22	30.8	37.4	34.7
Paralytic + encephalitic	51	28	79	38.2	32.9	36.3
Total	179	125	304	31.2	31.2	31.2
Fatal cases	10	4	14	52.2	32.0	46.4

The percentage of paralytic and encephalitic forms then increases with age, reaching 75% in the 71-80-year age-group, although the number of cases is too small for this to be highly significant. Apart from the first 10-year age-group, the severity of the disease thus increases in direct proportion to age. It may be that the relatively malignant course in the first 10 years indicated by these figures is at least partly illusory, since the cases included here were perhaps not of uniform etiology. As Grinschgl points out (see page 559), there is a possibility, judging from the neurological findings and apparently negative virological results, that some of the paralytic children were suffering from poliomyelitis.

In the 304 epidemic cases, the average age is the same for both men and women—namely, 31.2 years. For meningeal cases, the average is 29.4 years. For the paretic and encephalitic forms the average age is 36.3 years, but there is a distinct difference in the average age by sex, that for men being 38.2 years against 32.9 years for women. The average age of the 14 fatal cases is 46.4 years (see table III).

A similar age distribution is found in RSSE, as reported by Vizen, Filipovich & Shorina.[31] In poliomyelitis, on the other hand, the picture is appreciably different, with certain variations within individual epidemics; although the average age of patients has increased in recent years, possibly on account of changes in the character of the disease, the proportion of child cases is still 70%-90% and the average age 10-12 years.

Distribution of Epidemic Cases by Sex

Of our 304 patients, 179 were men and 125 women, a ratio of 1.4:1. Taking into consideration the possible modes of transmission of the virus, which will be discussed later, it would appear that men might be more prone to attack because of increased exposure to the sources of infection. In addition, predisposing and precipitating factors, particularly external factors and physical infirmities, probably play a bigger part in the contraction of the disease by men than by women. With poliomyelitis too, male patients predominate; according to Fanconi,[4] the average ratio of men to women is 1.3:1. However, since the great majority of poliomyelitis cases occur in children, factors other than those suggested in the case of virus meningo-encephalitis must be responsible for this difference in the sex distribution.

Distribution of Epidemic Cases by Occupation

It has already been seen from the discussion of the geographical distribution that the rural population was particularly affected in the 1953 epidemic; 237 patients (78%) came from rural areas, whereas only 67 (22%) came from the city of Graz or from large market centres or small towns. Of the first group, peasants, agricultural and forestry workers, woodcutters, and women working in rural households account for 157 cases (see table IV). The particularly high proportion of agricultural and forestry workers appears significant in view of the probable mode of transmission discussed below; a higher incidence in this occupational group is also mentioned in connexion with RSSE.[21] There is no special occupational distribution of cases in the urban population, but in most cases it was possible to find some connexion with a rural environment, for instance, a house at the edge of the city with a garden or a small farm, holidays in the country, or excursions in the wooded outskirts of the city. Consequently, a rural source of infection probably also plays some part in this population group. Whereas in the rural group men accounted for 147 cases and women for 90 cases, in the urban group men and women were approximately equally affected, with 32 male cases and 35 female cases. This seems to confirm our view that the higher incidence in males may be related to a correspondingly higher degree of exposure, for in the urban

TABLE IV. DISTRIBUTION OF 304 CASES BY OCCUPATION

Occupation	Rural population *		Urban population *	
	male	female	male	female
Agricultural workers and peasants	63	32	0	0
Foresters and woodworkers	26	0	0	0
Artisans	19	0	11	0
Factory and other manual workers	11	7	4	5
Employees and independent professions	7	4	12	2
Women in households	—	36	—	24
Schoolchildren (6-14 years)	17	9	4	4
Infants (2-5 years)	4	2	1	0
Total	147	90	32	35

* The ratio of cases among the rural population to those among the urban is 3.5 : 1.

group, where contact with rural sources of infection was accidental in the sense that it was not determined by occupation, an equal number of men and women were affected.

Possibility of Virus Transmission

As was to be expected in view of the assignment on virological grounds of this meningo-encephalitis to the group of arthropod-borne encephalitides, we found a history of insect bites in a large proportion of our patients. In more than one-third of the cases, tick bites had occurred more or less shortly before the onset of the disease; in most of these cases, the persons had been bitten while engaged in rural work, such as hay-making, harvesting, forestry, or woodcutting. Repeated tick bites, either on one day or over a period of time, were frequently reported. The intervals between the reported tick bites and the development of symptoms were, however, extremely variable, ranging from a few days to several weeks. It would therefore seem that the possible incubation period itself is very variable. However, the dates given for tick bites were often not very dependable and we feel that it is quite possible that peasants and agricultural workers were in fact bitten by ticks without noticing it while engaged in strenuous work. People living in the country said that in many regions ticks were more numerous in 1953 than they had been for a long time.

Curiously enough, some of the urban patients also reported that they had been bitten by ticks during country excursions, berry-picking, etc.

So far as numerical comparisons can be made, it seems that a positive history of tick bite was almost more frequent in the urban than in the rural group, but this may simply be due to the fact that such an event is more unusual for a city dweller and therefore more likely to be remembered.

Quite apart from the fact that there were variable intervals between the tick bite and the development of symptoms, however, there was a large group of patients for whom any contact with ticks could definitely be excluded, and this supports our view that other blood-sucking insects or arthropods must be involved in the transmission of the virus. Mosquitos, particularly, must be suspected. Many patients reported spontaneously that they had been bitten by mosquitos shortly before their illness, and many of our epidemiological investigations revealed stagnant pools, open drainage ditches, and small ponds in the immediate neighbourhood of the houses or farms of patients and serving as breeding-places for mosquitos. In some cases patients reported being bitten by mosquitos on bathing trips.

In any case, we feel justified in stating that in general the chain of infection by which the virus is transmitted to man is variable and may involve various arthropods. We still cannot state what the natural reservoir of the virus is, but, as is the case with RSEE and louping-ill, local fauna, such as domestic animals, game, and small rodents, should probably be suspected.

Although transmission by insects may be the rule, we believe that other forms of infection may occur on occasion. This belief is supported by a series of cases in families where infection from a family contact would seem probable in view of the manner in which the cases followed each other, and particularly since no insect bites were reported. One instance involving three members of the same family was especially impressive: a sawmill worker fell ill at the beginning of June 1953 with the meningeal form of the disease, his 21-year-old daughter became paralysed on 26 June and died on 1 July of ascending paralysis, and finally, the younger daughter was admitted to the clinic with mild meningitis on 19 July. On the basis of reported cases of laboratory infection with louping-ill,[23, 24, 33] Olitsky & Casals [21] considered that infection from droplets or dust is possible in the arthropod-borne encephalitides. Similar laboratory infections have also occurred with the virus of the Czech tick encephalitis as reported by Gallia, Rampas & Hollender [5] and by Molnar & Fornosi,[20] and with the RSSE virus as reported by Jervis & Higgins.[12] In rare cases, transmission through foodstuffs may also be possible; three of our patients, all with the meningeal form of the disease, regularly drank milk from the same goat. We found this particularly interesting, since Shtilbans,[27] in a paper on the possibility of acquiring tick encephalitis without contact with ticks, suggests that the disease can be transmitted in raw milk from infected goats.

Predisposing and Precipitating Factors

As is the case with many diseases, external factors probably play some part in determining the symptoms and severity of virus meningo-encephalitis. We frequently found that our patients had engaged in strenuous physical activity shortly before the onset of disease: peasants and agricultural workers had overexerted themselves during harvesting; manual workers and labourers had done particularly heavy work; other patients had undertaken fatiguing journeys. Some patients reported that they had become soaking wet and had then had to walk long distances in wet clothes. Patients had very often been exposed, while bathing or while working out of doors, to strong sunshine for a long time, and the doctors who referred these cases to us consequently thought at the onset of the illness that they were dealing with cases of sunstroke. Minor accidents, falls, or work injuries were sometimes reported to have occurred a few days before the onset of symptoms. In one case a tonsillectomy, and in another an appendectomy, had preceded the disease; in the latter case, symptoms of tetanus developed during the meningitic stage, and the patient died. It is possible that the appendectomy had afforded an opportunity for virus meningo-encephalitis to manifest itself, and that the period of incubation and the severity of the tetanus infection, which probably followed the operation, were influenced by the virus infection. On one occasion a meningo-encephalitic form of the disease developed when serum sickness following a tetanus antitoxin injection had subsided. Three of our patients were women in the fourth to the ninth month of pregnancy.

So far we have no information concerning endogenous predisposing factors.

The external factors mentioned above were acting—where the disease clearly had a diphasic course—partly before the onset of the initial stage and partly during the latent period. It therefore seems quite possible that in many cases the disease would have been abortive but for the effect of external factors during the latent period, and that there would have been no invasion of the central nervous system. External factors may also play a part in determining whether the disease follows a mild meningeal course or results in severe encephalitic or paralytic symptoms. As in poliomyelitis, very heavy strain on certain muscle groups may also influence the localization of paralysis. One case is significant in this connexion : a peasant girl, who was already feverish and suffering from pain in the left arm, carried heavy loads with that arm for several hours; on the following day paresis of the left shoulder-girdle and upper arm set in.

We were unable to find any direct influence of weather on the appearance of the disease. However, it did seem to us that the number of new cases decreased somewhat during certain rainy periods during the summer, only to increase again with the return of fine weather. The explanation of this

would seem to be that during periods of bad weather persons in whom the disease is already latent are less exposed to such external factors as strenuous work on the land or exposure to strong sunshine, and that symptoms appear only when these factors become more pronounced with the return of the fine weather. Thus there seems to be a lengthened incubation period or quiescent stage.

So far we have no information on the excretion of the virus from the human body and on its subsequent fate. We shall carry out further investigations on this point and on the virus reservoir. The possible modes of transmission, the portals of entry, and the length of the incubation period have also still to be clearly determined.[a] It is to be hoped that these problems will be successfully solved, providing a working basis for effective prevention of this disease.

RÉSUMÉ

L'étiologie de plusieurs des méningo-encéphalites à virus, dont l'incidence s'est accrue considérablement ces dernières années, demeure obscure. Il est par conséquent difficile de comparer les épidémies survenues en diverses régions de l'Europe centrale. On sait pourtant qu'il existe des zones d'endémicité où le nombre des cas observés a pris en certaines années des proportions épidémiques. La région de Wiener Neustadt par exemple, au sud-ouest de Vienne, est connue depuis vingt ans comme foyer permanent de méningo-encéphalite. Des cas ont été signalés aussi dans la zone de la Carinthie voisine de la Yougoslavie. Les méningo-encéphalites ont été fréquentes également dans la Styrie orientale, qui a connu aussi une morbidité élevée par poliomyélite en 1947.

L'épidémie de méningo-encéphalite survenue en Styrie en 1953 et celle qui éclata en Slovénie au même moment ont certainement une origine semblable. L'étude de l'épidémie de Styrie, qui fait l'objet de cet article, se rapporte uniquement à 304 cas hospitalisés à la Clinique neurologique de Graz en 1953 (des centaines d'autres cas ont été soignés dans les hôpitaux régionaux). L'épidémie montra une périodicité saisonnière très nette: augmentation du nombre des cas durant la période de juin à septembre, avec déclin durant les mois d'hiver, le maximum survenant au cours des 29e et 30e semaines de l'année. Le pourcentage de cas paralytiques et encéphalitiques le plus bas (20% du total des cas) a coïncidé avec l'apogée de l'épidémie. Cette proportion s'est élevée à 48% en octobre-novembre, ainsi qu'au début de l'épidémie. La morbidité a été plus élevée dans les zones rurales que dans les villes. Ce fait a été relevé également dans d'autres provinces de l'Autriche, en Bohème et en Moravie.

Les groupes d'âge inférieurs ont été relativement peu affectés. Seize seulement des malades observés (environ 20% du total) avaient entre 1 et 10 ans et la maladie ne fut fatale à aucun d'eux. La morbidité maximale (48% des cas) s'observa dans le groupe de 11 à 30 ans — et plus tôt chez les hommes que chez les femmes. La proportion des formes paralytiques qui est de 38% environ chez les enfants de 1 à 10 ans, augmente avec l'âge.

[a] Since this paper was submitted for publication (August 1954), further epidemiological investigations, which are still in progress, have yielded certain additional details regarding the virus reservoir and possible modes of transmission. These investigations will be reported on when complete.

Le virus est transmis par des arthropodes, par les tiques et probablement les moustiques. Les modes de transmission du virus à l'homme semblent divers: plusieurs espèces d'arthropodes peuvent être en cause, ainsi que des poussières ou le lait de chèvres infectées, comme le suggère un certain nombre d'infections familiales.

Le surmenage, une exposition excessive au soleil, des traumatismes, des injections, des opérations chirurgicales ou la grossesse semblent prédisposer à la maladie. Il est probable aussi que des facteurs externes exerçant leur action durant la période prodromique ou la période de latence de la maladie en aggravent le cours.

Pour préconiser des mesures prophylactiques efficaces il faudrait établir par quelles voies le virus pénètre dans l'organisme et en est éliminé; il faudrait connaître la durée de la période d'incubation et savoir quel est le réservoir du virus dans la nature.

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