NEUROPARALYTIC ACCIDENTS COMPLICATING ANTIRABIC TREATMENT

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The occurrence in Palestine within the three-months period April 28th to July 29th, 1932, of three paralytic accidents following on treatment with a killed carbolized rabies virus vaccine is without precedent or parallel anywhere, and would therefore appear to merit record, description, and discussion. In the series are included two fatally ending cases of Landry's paralysis and one case of paresis of the lower extremities in which recovery took place without loss of control at any time over the urinary and rectal sphincters. The fact that on this occasion an opportunity was afforded of studying in the central nervous system the pathological anatomy of an exceedingly rare condition, of which the aetiology still remains obscure, lends added interest to the occurrence.

CASE I

This was a young Moslem, aged 29 years, whose family history recorded no affections of the nervous system, and no evidence of mental disease, paralysis, or fits. There was nothing in the patient's occupation, environment, or mode of life likely to injure health or predispose to nervous instability. He had at no time suffered from any acute illness or infectious disease, and there existed neither chronic nephritis nor abnormality in the circulatory system. For a man of his class he was of more than average intelligence.

On April 13th, 1932, he was severely bitten by a camel, which inflicted, through the clothing, a deep wound on the right upper extremity. Antirabic treatment was begun on April 15th, but, on the camel's remaining healthy after ten days' observation, was discontinued on April 26th. During the period of vaccine administration the patient's conduct was exemplary; no local accidents (or accidents with a local point of departure) occurred, and he showed no undue signs of nervousness. On April 28th, without any premonitory fever, headache, vomiting, or backache, he experienced a feeling of weakness in all four extremities. On April 29th a progressive loss of power in arms and legs was noted, but he was still able to move about, although with difficulty. Next morning an almost complete motor paraplegia was found to have replaced the earlier paresis; he was unable to walk or to raise his arms, and in the afternoon he was brought to the Government Hospital in Jerusalem.

Examination revealed a total flaccid motor paralysis of the extremities. Reflexes, both superficial and deep, were abolished. Cutaneous sensibility was perfectly intact. There were no changes in the nerves of special sense. Both pupils reacted to light and to accommodation, and were of equal size. Intelligence and memory were unimpaired. The bladder and rectum were unaffected. The legs and arms could be moved passively without muscular resistance, and the muscles showed neither active nor reflex tension. The pulse rate was 90, the respiration rate 22. The patient's condition remained unaltered till the afternoon of May 3rd. Then, although the temperature remained normal, the pulse rate was 150, the respiration rate 40, and breathing was laboured. The neck muscles were paralysed. There was retention of urine and faeces. Dysphagia and dysarthria were present as well as certain ocular symptoms—namely, marked dilatation of the left pupil, hippus, and ophthalmoplegia interna. No changes in the fundus oculi were seen. Blood pressure was 128/90. White and red cell counts were respectively 18,700 and 5,400,000 per c.mm., and the differential count showed the percentages of leucocytes to be: polymorphs 85, lymphocytes 14, eosinophils 0.5, and basophils 0.5. The spleen and liver were not palpable. Muscular wasting had not occurred, and the electrical reactions were normal. The urine contained

a trace of albumin. Complaint was made of a feeling of oppression in the chest.

On May 4th the disease had progressed; patient was drowsy and unable to talk or answer questions; the pulse was rapid and almost imperceptible; the respirations were shallow and slow; face and fingers were deeply cyanosed. Death took place, after an illness of six days' duration, at 1.45 p.m. A post-mortem examination was made at 4 p.m. on the same day.

CASE II

On July 13th, in a Jewish settlement in Southern Palestine, a dog, suspected of rabies, was reported to have bitten several other dogs, including one which belonged to the family of the case about to be described. In spite of the fact that no member of the family had been bitten by the dog and that all the animals concerned were destroyed on July 16th, antirabic treatment was requested from the medical officer of the settlement on behalf of the patient—a boy of 5 years—whose mother was 30 years old and whose sisters were respectively 9 and 2.

Whereas, however, the mother and sisters received a complete course of treatment without untoward incident, the boy developed, after the tenth injection, symptoms of an acute ascending spinal paralysis. The patient was a Jewish boy of Bulgarian extraction. His family history was without evidence of nervous disease, but he himself was an exceptionally highly strung child. He had never contracted any infectious disease and there had been no occurrence of medical interest during his life. Antirabic treatment was begun on July 19th and continued till July 31st, when the boy's condition precluded further injections. On July 29th complaint was first made of pains in the feet and legs, and it is noteworthy that the onset of symptoms happened immediately after the patient had taken a cold shower-bath and three days after he had been severely punished by his father. By July 31st the legs were completely paralysed and pains were felt in the hands and arms. On August 3rd the patient began to experience difficulty in swallowing and in breathing, and on that day he was sent to the Government Hospital in Jaffa.

Examination revealed a complete flaccid paralysis of both legs, with abolition of the deep tendon reflexes. No response was obtained on stroking the outer margin of the sole of the foot and no muscular contraction on striking the periosteum and fasciae. Cutaneous sensibility remained unchanged, and control over the urinary and rectal sphincters was preserved. The abdominal reflexes were absent. There was partial paresis of both upper extremities and a paralytic condition of the sixth right cranial nerve. Pupillary reactions to light and accommodation were normal. The patient could articulate distinctly, but was unable to swallow solid foods. Tongue movements were unimpaired. Respiration was forced and deep; the temperature varied from 37° C. to 37.5° C.

On August 4th the general condition was little changed, but a definite paresis of the right facial nerve was now marked. During the course of the evening paralysis of both arms became total; Cheyne-Stokes breathing supervened, and the patient was no longer fully conscious. On August 5th the temperature rose to 38° C. and death ensued at 9 a.m. A post-mortem examination was carried out at 10 a.m. on the same day.

CASE III

The patient was a Jewish girl of Palestinian origin, and was at the time of the occurrence 5 years of age. She had never suffered from any infectious disease, and physical examinations, made during and shortly after her period of vaccine treatment, revealed no constitutional abnormality. In the left eye there was marked internal strabismus, but she was in every respect a well-developed healthy child. Although she had not recently experienced any trauma or chill, she had, it may be noted, been bitten by a dog in 1931, and had then undergone a short course of antirabic treatment. On that occasion but eight injections had been administered as the biting animal proved to be healthy after ten days' observation. No ill-effects had followed the treatment. The family history negated the idea of any predisposition by heredity to nervous disease in the child.

On June 28th the patient was scratched—not bitten—by a cat near Haifa, a town in Northern Palestine—where antirabic

treatment was commenced at her parents' request and continued for only ten days, the cat having remained healthy. On July 11th the child did not feel well, and on the next day complained of weakness in the legs when she tried to walk. On July 13th she developed, near the sites of inoculation, an urticaria, which was accompanied by general malaise, nausea, and vomiting. Later in the day she found it impossible to stand up, and was transferred to a private hospital in Haifa. Clinical investigation at this time showed normal movements of hands and arms, weakly positive abdominal reflexes, no evidence of cerebral disturbance or pupillary change, and no involvement of bladder or rectum. In the legs there was no muscular atrophy or appreciable lowering of muscle tone; passive movements were fully possible, and active though very feeble movements of all joints could be elicited. The patellar and Achilles reflexes were abolished, but the cutaneous reflexes were intact. No extension response to Babinski's reflex was obtained. A generalized itching and a disagreeable cough contributed to the child's extreme irritability.

On July 15th the urticaria and itching became more pronounced and the ataxia more definite; otherwise the condition remained unchanged. There was no motor weakness in either arm, no disturbance of bladder or rectum, and no obvious alteration in cutaneous sensibility. On July 17th, although the paresis in the lower extremities showed no improvement, the electrical excitability of the muscles was undiminished; the urticaria and itching became less troublesome and the cough easier. By July 19th the patient's state was considerably better: she could sit unsupported in bed for short periods, but not yet stand up; the itching, urticaria, and cough had disappeared. On August 21st a further improvement was noted: the leg movements were more forcible and the ataxia was hardly discernible; the patient could now stand up and walk a few steps with support, but tired quickly. Electrical reactions were normal. Within ten days recovery was complete and the child was discharged from hospital.

SPECIFIC TREATMENT

In Palestine the vaccine employed is a 2 per cent. emulsion of fixed-virus brain, killed by the combined action over twenty-four hours of heat at 37° C. and of 1 per cent. phenol, and diluted to half with physiological saline solution prior to use. Ordinarily, treatment consists of the daily administration, over fourteen days, of 5 c.cm. of this killed carbolized virus, but in none of the cases under review was the full course given. Cases I and III received only ten injections, Case II twelve. Paralytic accidents followed, therefore, on the introduction of 0.5 gram and 0.6 gram respectively of nervous tissue.

TYPES OF ACCIDENT: DIAGNOSIS: MORTALITY RATES

Cases I and II presented an almost identical pathological condition, in which the main feature was a rapidly advancing, flaccid paralysis, without accompanying fever, without sensory disturbances, without loss of sphincter control, and terminating fatally within a short time from involvement of the bulb. The clinical picture is that of Landry's syndrome, but an affection of cranial nerves in both cases is worthy of remark—in Case I paralysis of the oculomotor, in Case II paralysis of the abducens and facial. Remlinger (1927), however, insists upon the frequency of the involvement of the facial nerve in the paralytic accidents of antirabic treatment. Case III, in which paresis of the lower limbs occurred without bladder and rectum troubles, was, in the neurologist's opinion, a very mild type of dorso-lumbar myelitis.

Clinically, paralytic accidents show the greatest diversity. When, however, classification is based on their degree of intensity, they are capable of division into three main types: neuritis forms, or simple localized paralytic referable to lesions of peripheral nerves; dorso-lumbar myelitis; and paralysis of the Landry type. Avezzu (1923), from his analysis of 140 cases, has found the

relative frequency and mortality of these groups to be as follows: neuritic forms 35 per cent., without fatality; dorso-lumbar myelitis 30 per cent., with a mortality rate of 5 per cent.; paralysis of the Landry type 35 per cent., with the high death rate of 30 per cent. The relative incidence given by Avezzu, however, differs considerably from that estimated at the International Rabies Conference of 1927. Then it was shown that of 243 cases of paralysis reported from different antirabic institutes only thirty-nine (or 16 per cent.) presented the Landry syndrome. Again, within our own experience in Palestine, five cases have occurred since 1928; of that number all three showing Landry's paralysis proved fatal, while the remaining two with dorso-lumbar myelitis recovered.

ONSET OF SYMPTOMS IN RELATION TO PROGNOSIS

In Case I the patient, bitten by a healthy animal, showed first symptoms thirteen days after the commencement of treatment and fifteen days after the date of bite. In Cases II and III, in which the patients were not bitten at all, symptoms appeared respectively ten days and thirteen days after the first antirabic inoculation. Paralytic accidents usually occur within thirty-five days of commencement of treatment. The greatest incidence falls within the first three weeks, and the earliest date of onset yet recorded is the fifth day (Braut, 1897). The most serious cases appear during the second week of treatment—that is, from the seventh to the twelfth day; in the milder forms onset is commonly delayed until the end of treatment, or after its termination. These observations are of considerable value in prognosis. A study of Cases II and III makes it abundantly clear that there is no connexion between the date of bite and onset of symptoms.

INCIDENCE

Present knowledge of incidence has been largely derived from a series of records collected and published by several observers. Remlinger (1905) described forty cases among 197,712 patients treated, Babes (1912) eight among 6,525, Simon (1913) 103 among 211,744, Pelsler (1920) 200 among 260,000. At the Rabies Conference of 1927 it was shown that since the introduction of Pasteurian treatment in 1885, there had occurred among 1,164,264 inoculated patients 329 cases of paralytic accident, while more recently McKendrick (1930) has reported a further eight among 31,656 persons treated during the period 1926–8 at various institutes. The incidence per thousand treated patients, therefore, varies considerably: according to Remlinger it is 0.37, to Babes 1.3, to Simon 0.48, to Pelsler 0.77, to the findings of the Rabies Conference 0.28, to McKendrick's statistics 0.25. Variation is, presumably, to be explained by lack of uniformity in statistical standards, but, if we accept as most reliable the two rates given last, it proves that a relatively rare disease is being discussed. Its rarity, however, by no means relegates it to a position of merely academic interest and importance.

Undoubtedly certain influences have an important bearing on incidence. The extremes of life are usually held to be immune. Thus among children, who constitute 50 per cent. of the total treated, paralysis has never been noted in the Pasteur Institutes of Egypt or of the U.S.S.R. The statistics of the Rabies Conference also show that among 149 cases in which the age was specified accidents did not occur below the age group 6–10 years. Yet the age at which Cases II and III of our present series developed paralytic symptoms was only 5 years—the earliest age so far recorded. An almost equal liability to accident is observed in the two sexes, but the condition is more frequently encountered among Europeans than among indigenous races of warm countries. There is

a preponderance of cases among those engaged in intellectual pursuits as compared with purely manual labourers. There is no evidence that season affects incidence in any way; cases crop up at any time during the year without apparent reference to month or meteorological variation; on the other hand, there exists an undoubted, although as yet unexplained, periodicity about this disease exactly comparable with that sudden increase in incidence recorded from time to time in the encephalomyelitis following or complicating certain of the exanthemata (measles, variola, vaccinia, typhoid fever, etc.). The question as to whether a person has been bitten by a rabid or by a healthy animal, or indeed has not bitten at all, does not appear to be of importance in the production of paralytic accidents. A study of their occurrence in relation to the date of the bite and to the date of the commencement of treatment must lead to the inevitable conclusion that the sole prerequisite of accident is that treatment has been administered. But, whereas the appearance of paralysis is independent of the nature of the biting animal, the method of treatment and individual predisposition most certainly play predominant parts in causation. Moreover, it would seem to have been sufficiently established that the protective inoculations themselves are ultimately responsible, and that no matter what modifications of Pasteurian treatment have been employed, no matter to what degree the basic nerve substance of antirabic vaccines must thereby have been altered—whether by chemical or physical means—accidents have surely, even if rarely, supervened.

The proportion of cases of paralysis to the number of treated persons, however, varies markedly according to the method of vaccination employed, and in this connexion the statistics of the Rabies Conference proved conclusively the superiority of carbolized vaccines over all others. McKendrick (1930) also, from an analysis of the data furnished by thirty-one Pasteur institutes during the period 1926–8, has shown that there occurred eight accidents among 31,658 patients treated; of this number, however, dried cords accounted for five cases out of 2,318 treated, dilution methods for two out of 6,362, and phenol vaccines for one out of 17,538. Indeed, the degree to which paralysis has complicated treatment with killed, carbolized virus up to the present day is reflected in the fact that there are on record only sixteen authentic cases: at Lemberg, one accident among 2,213 persons treated—an incidence rate of 0.45 per 1,000; at Jerusalem, five among 9,959, or 0.5 per 1,000; at Kasauli, two among 7,293 Europeans and one among 77,548 Indians, or 0.27 and 0.013 respectively per 1,000; at Lwow, one among 2,313, or 0.43 per 1,000; in the U.S.A., six among 17,645, or 0.34 per 1,000. It is, however, important to recall that in Palestine the first accident occurred after 4,580 bitten persons had been treated, the second after 5,409, and the fifth after 9,950. Moreover, during 1932, three cases occurred within three successive months, when only 320 patients had attended for antirabic inoculations. Again, while the period 1923–7 was entirely free from accident, the period 1928–32 showed five cases of post-treatment paralyses. To this, however, a striking parallel is to be found in the statistics of the Vienna Institute: whereas, between 1894 and 1914, 7,632 bitten persons were treated without accident, between 1915 and 1923, 6,764 treatments yielded thirty-nine cases (5.7 per 1,000).

These figures lend support to the view that the incidence of paralytic accidents shows periodic increases which, however, neither in Palestine nor in Vienna, can be ascribed to intensification of treatment or be explained by the number of passages to which the fixed virus used for vaccine purposes has been submitted (Bujwid's theory). Finally, in the light of our recent experience in Palestine,

it has to be admitted that the safeguards afforded by the use of carbolized vaccines are far from absolute.*

PATHOLOGICAL ANATOMY AND HISTOLOGY

To quote Remlinger (1927): "The pathological anatomy of the paralyses of treatment is imperfectly known. The first Rabies Conference added nothing to our knowledge of the subject. Deaths are, as a matter of fact, very rare, and a post-mortem examination is often refused by the families of the deceased." The last observation fully accounts for our present lack of precise information. Moreover, perusal of the relevant literature shows the macroscopic appearances in brain and spinal cord to vary considerably.

Babes and Mironesco (1908—one case): oedema of brain, cord, and investing membranes; marked softening of the cord, especially in the lower dorsal and lumbar segments; differentiation between white and grey matter no longer possible; the cord surface of a homogeneous greyish-pink appearance with loss of typical design. J. Koch (1912—one case): localized oedema of the pia in the region of the lumbar enlargement; serous meningitis, and small areas of softening in the lumbar cord. Simon (1913), in an analysis of the reports on twelve necropsies: hyperaemia of the central nervous system; intense myelitis with, specially in the lumbar swelling, destruction of the white substance of the cord. Jochmann (1913—one case): meningitis serosa circumscripta in the cervical cord; meninges, over the lumbar portion, oedematous. Fielder (1915—one case): acute softening in the cord, most pronounced in the cervical region, least severe in the lumbar segment. Koritschoner and Schweinburg (1925—seven cases): (1) haemorrhagic myelitis; (2) disseminated myelitis, with destruction of the white matter in certain areas; (3) general congestion and oedema in cord; (4) transverse myelitis; (5) intense diffuse myelitis; (6) severe myelitis; oedema, congestion, and haemorrhage; similar changes in brain; (7) general hyperaemia. Smith and Murphy (1930—one case): vessels throughout the brain markedly congested; brain and cord grossly negative.

It will be seen that invariably the lesions described are of a totally different order from those of rabies. Negri bodies are never present; passage to rabbits produces no symptoms; lesions are distributed uniformly through white and grey matter alike, with a preponderance in the dorso-lumbar cord.

FINDINGS AT NECROPSY

In our two fatal cases of Landry's paralysis, permission to investigate the central nervous system was fortunately secured, and these partial necropsies were performed with the least possible delay in order that the occurrence of post-mortem changes might be obviated. Both cases showed an almost complete absence of gross pathological alterations. No inflammation, oedema, swelling, or areas of softening obtained, no obvious destruction of the white matter in brain or cord. There was no loss of design in any part of the spinal cord, differentiation between white and grey matter being clearly marked. The meninges were unaffected. In Case II the vessels were somewhat congested, and here and there throughout the central nervous system small haemorrhages were noted.

No positive results followed the direct subdural inoculation of rabbits with emulsions of either brain. The microscopical examination of nervous lesions in fatal cases of paralytic accident has been, perhaps, too infrequently undertaken to assign as yet to the condition any definite histological criteria. On the other hand, the minute changes described by Babes and Mironesco (1908), Jochmann (1913), Fielder (1915), and Bassoe and Grinker (1930) would seem to establish the histological identity of such paralyses with the nervous disorders occasionally complicating certain of the exanthemata (small-pox,

* The carbolized vaccines productive of the various accidents were in each case fully tested on rabbit series with entirely negative result.

vaccinia, measles, varicella, and typhoid)—the occurrence in the central nervous system of perivascular zones of demyelination together with some degree of perivascular cuffing—that is, infiltration of the adventitial lymphatics with cells of the lymphocyte class. Moreover, should this same microscopical picture, so highly characteristic of the condition known as acute disseminated encephalomyelitis (Westphal), be constantly present in cases of paralytic accident, then antirabic treatment must necessarily be added to the somewhat heterogeneous group of factors (small-pox, vaccinia, measles, etc.) capable of producing that condition. Indeed, its inclusion within the group has already been suggested by Perdrau (1928), Greenfield (1930), and Marsden and Hurst (1932).

It is not our intention to give here any detailed account of the histology of the two fatal cases in our present series. For the sake of completeness, however, permission to include a summary of the principal findings has been secured from Professor S. Getzowa, formerly of the University of Bern, who has aided us in our recent investigations and in collaboration with whom we hope shortly to publish elsewhere a contribution to the histology of Landry's paralysis. In the nerve centres the brunt of the attack was borne by the ganglion cells, in which, varying with the region examined, all stages of degeneration were observed, from slight chromatolysis to nucleolysis and complete necrosis. Periganglionic and perivascular cell infiltrations did not occur, and perivascular zones of demyelination were wholly absent. Axis cylinders and myelinated sheaths were everywhere unaffected. Neuroglia was not increased. Fat stains proved negative, but in Case II, in certain areas above the medulla, a fatty degeneration, strictly limited to the walls of the vessels, was not infrequently observed. The small vessels of the central nervous system showed little or no narrowing of lumen or thickening of the walls; they were, however, much congested, and haemorrhages were not infrequent. The condition was essentially an intoxication in contradistinction to an inflammation. In the spinal ganglia the capsule showed cellular proliferation; most of the nerve cells here had undergone necrosis, and several had been replaced, in whole or in part, by endothelial cells.

AETIOLOGY

Remlinger (1927) has already shown it to be unnecessary to seek an explanation of the occurrence of paralytic accidents in fixed virus, street virus, street virus modified by treatment, rabies toxin, enzyme or ferment action, bacterial infection, anaphylaxis, neurotropic virus, or *génie épidémique neurotrope*. Marinesco (1908) attributes these paralytic accidents to a poison of normal nervous tissue, and we (1928, 1930), in support of this cytotoxin theory of origin, have stated that "the cause lies in the antirabic injections themselves, and more specifically in some component of the nerve substance necessarily introduced during the process of immunization." To this view we still adhere although, admittedly, had the histology of our two recent cases proved to be identical with that of the four cases described by previous writers and therefore with that of acute disseminated encephalomyelitis (Westphal) or of acute perivascular myelinoclasia (Marsden and Hurst), we might have thought fit to revise our position and subscribe to arguments favouring an encephalomyelitic virus as *causa causans*.

SUMMARY

1. Paralytic accidents are a rare complication of antirabic treatment, but exhibit a certain periodicity in their occurrence.

2. Since 1928 the condition has been observed in Palestine on five occasions: three patients showing Landry's syndrome have died, two with dorso-myelitis have recovered.

3. Incidence is now, for the first time, recorded among members of the age group 0-5 years.

4. The proportion of cases to the number of treated persons varies according to method of vaccination employed. In this connexion, statistics have proved the superiority of carbolized vaccines over all others, but now the safeguards thereby afforded are shown to be far from absolute.

5. The histopathology now described does not agree with that previously published. In our cases the microscopical appearances of brain and spinal cord revealed little more than varying degrees of degenerative change in the nerve cells, together with considerable vascular congestion and occasional haemorrhage. Evidence of perivascular demyelination or of perivascular cuffing was altogether wanting and, therefore, the type of paralytic accident represented by the cases under review cannot, on histological grounds, be included among the group of diseases termed "acute disseminated encephalomyelitis" or "acute perivascular myelinoclasia." Further, the absence of Negri bodies and of perivascular and periganglionic infiltrations differentiates the condition from rabies.

6. A cytotoxin, rather than a virus theory of origin, is again advanced. Thus, in the basic nerve substance of all antirabic vaccines there seems to exist some deleterious component which, though adversely affected by various physical and chemical agencies, is still capable, in peculiarly susceptible individuals, of producing neuro-paralytic disorders.

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THE WORKING CAPACITY OF THE CONSUMPTIVE

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It is not often that tuberculosis workers are in a position to correlate clinical findings, sociological environment, and precise economic status in patients under their care in the exact way in which this is possible in a village settlement for the tuberculous. At Preston Hall there are just over 150 settlers—all ex-patients—living in the village and employed in the different industries attached to the sanatorium section. From this number ten men have been selected for the purpose of this note. I believe that a detailed statement of the known working capacity of a selected group of patients with positive sputum may interest those who so frequently, and often with good cause, cast doubts on the ability of the patient with relatively advanced pulmonary tuberculosis to earn his livelihood.

CASE I

The patient, a male aged 36, left school at the age of 14, and served for seven years in the machine-tool trade. In 1917 he joined the Flying Corps, but after eleven