INFECTIOUS NEURONITIS

A REVIEW OF THE LITERATURE

BY

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Neuritis has been defined (6) as a "painful, degenerative (often inflammatory) process in any part of the peripheral neuron". In this sense the term is generic, including anterior poliomyelitis, at least insofar as the disease affects the anterior horn cells, neuronitis, radiculitis and peripheral neuritis. Moreover, the more recent pathologic studies have made clear that when either the cell body or the peripheral process is affected the other is at the same time diseased.

Since the war, special attention has been paid to a form of generalized polyneuritis which is apparently infective. It has been variously referred to as the Guillain-Barre syndrome (17), polyradiculoneuritis (1, 15), infective polyneuritis (3, 25), and infective neuronitis (13, 14, 18, 19, 27, 29, 34). Whether or not the conditions called by these various names are a disease entity, separate diseases, or simply varied manifestations of a single underlying process remains for the future to decide.

HISTORICAL

Sir William Osler (24) in the first edition of his Textbook of Medicine, published in 1892, first described the condition under the title "Acute Febrile Polyneuritis." Mills in 1898 (21) originated the term neuronitis as descriptive of a process involving the entire peripheral neurone. This term was reintroduced by Kennedy (14) and has since found more or

less general acceptance. Laurans* reviewed the literature to 1908 and collected cases dating back as far as 1869. However, it was not until the World War that any thorough study was made. Guillain, Barre and Strohl in France (11), and Bradford, Bashford and Wilson (3) and Kennedy (14) in England reported a total of thirty-six cases seen at the front, with careful pathologic and etiologic studies. Since that time, sporadic cases have been reported from the United States and Great Britain.

THE CLINICAL PICTURE

Incidence:

A total of 110 cases has been collected from the literature of Great Britain and the United States. The literature of continental Europe includes many more cases which have not been reviewed. Of 60 cases in which age and sex were reported, (excluding the 36 cases reported from the army during the war) 37 were male and 23 female, a ratio of 1.6 to 1.0. The age distribution by decades is given in Figure 1. Attention may be called to the fact that 41 or 68% of the cases occurred in the three middle decades of life.

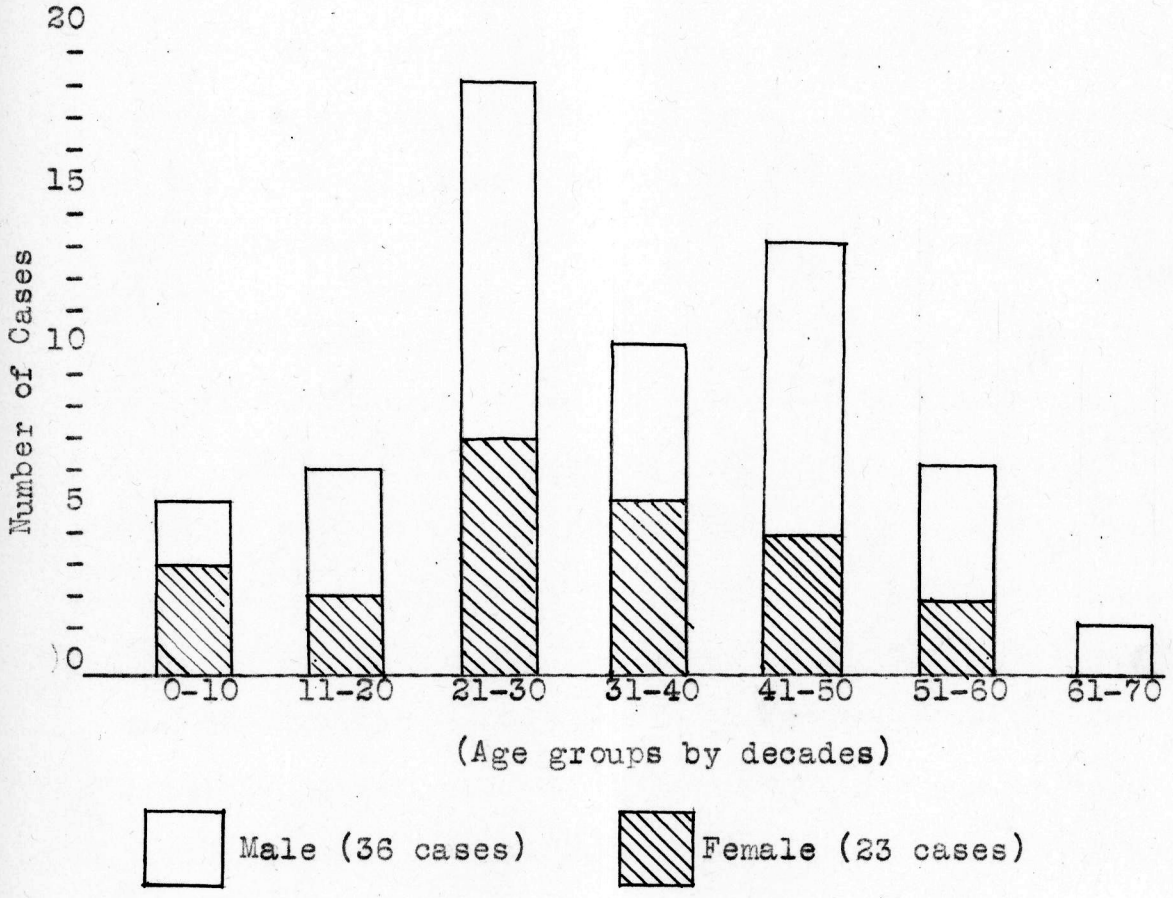
Onset:

Two modes of onset have been noted in reported cases. In the majority there is a definite history of illness with general symptoms preceeding the onset of the palsy. In the remainder the first symptom is neurogenic in character: weak-

* Laurans, A., These de Paris, 1908, p. 156; cited by Taylor and McDonald (32).

FIGURE 1

SHOWING AGE AND SEX DISTRIBUTION OF 110 CASES



ness, pareathesias or pain.

Osler in his original description of the disease (24) states that "the attack sometimes follows exposure to cold or overexertion, or in some instances comes on spontaneously. The onset resembles that of an acute infectious disease. There may be a definite chill, pains in the back and limbs and joints so that the case may be thought to be acute rheumatism. The temperature rises rapidly and may reach 103 or 104 degrees. There are headacke, loss of appetite and general symptoms of acute infection."

A history of preceeding febrile illness was obtained from 49 of 76 reported cases in which such a history was sought.* Guillain (10) does not mention it. Holmes (12) states that "almost all patients have stated that it began with general malaise...or...fever. In some patients the temperature did not exceed 100 degrees, but in others it rose to 1o2 and 103 degrees Fahrenheit." In Bradford's cases (3) the initial illness was characterized by moderate fever - 100 to 101 degrees, headache, and pain in the back. Occasionally there was sore throat; rarely merely catarrh.

The diagnosis at this time is usually of a cold or "flu", and the acute symptoms disappear after two or three days. Following this there is a latent period varying from

* See table 2.

TABLE 2

	Name Given Disease	Total Cases	Prec. Illn. +	Prec. Illn. −	Facial Paral. +	Facial Paral. −	Cran. Nerve +	Cran. Nerve −	Sphin. Dist. +	Sphin. Dist. −	Spinal Fluid Protein Inc.	Spinal Fluid Protein Nor.	Spinal Fluid Protein Unrec.	Outcome Recov.	Outcome Died	Outcome Unrec.
Guillain (11)	Radiculo-neuritis	10	(a)		2	8	1	9	2	8	9	0	1	10	0	0
Kennedy (14)	Infect. Neuronitis	4	4	0	4	0	2	2	3	1	0	1	3	0	1	3
Cassamajor (5)	Acute Infect. Meningomyelo-neuritis	2	1	0	1	1	0	2	0	2	0	0	2	0	2	0
Holmes (12)	Acute Febrile Polyneuritis	12	(a)		(b)		3	9	(c)		0	0	12	10	2	0
Bashford et al.(3)	Acute Infect. Polyneuritis	30	15	5	(d)		(a)		1	1	0	0	30	22	8	0
Viets (35)	Acute Polyneur. c̄ Facial Dipleg.	2	1	1	1	1	0	2	1	1	1	0	1	1	1	0
Strauss & Rabiner(31)	Myeloradiculitis	7	4	3	0	7	1	6	3	4	0	0	7	7	0	0
Taylor & McDonald (32)	Polyneuritis c̄ Facial Diplegia	16	8	7	16	0	3	13	5	10	5	1	10	11	3	2
Barker (1)	Acute Diffuse Polyradiculo-Neuritis	1	0	1	1	0	1	0	0	1	0	0	0	1	0	0
Pinckney (26)	Acute Infect. Polyneuritis	5	3	2	3	2	3	2	1	4	4	0	1	4	0	1
Gilpin et al. (8)	Polyneuritis "Neuronitis"	20	13	7	7	12	0	20	0	20	20	0	0	11	5	4
Madigan & Marietta (15)	Polyradiculo-Neuritis	1	0	1	1	0	1	0	0	1	1	0	0	1	0	0
	(Totals)	110	41	27	35	31	15	56	15	51	40	2	68	78	22	10

(a) Not Given (b)"Invariably" (c)"Almost Constant" (d) "Almost always".

three days to six weeks before onset of neurogenic symptoms. In view of this wide range in the length of the latent period, one may well doubt the relationship of the original febrile illness with the subsequent neuritis. Yet in other diseases, notably rheumatic fever, a similar condition obtains and the relationship is undoubted.

Motor Symptoms:

The paralytic stage occasionally begins with a general weakness, gradually progressing to complete paralysis. Usually however, the onset is more sudden and dramatic, the patient falling down or discovering that he can not rise from a sitting posture. The paralysis is widespread, affecting more especially the large muscles of the limbs and trunk but not exclusively limited to them. It is symmetrical, but the degree of involvement on the two sides is not always equal. Usually the leg muscles are first involved and with progression the picture may be that of a Landry's ascending paralysis. Less frequently the arms are affected first and the paralysis progresses downward (35). In a few cases muscular weakness, or diminished deep reflexes may be the only motor symptom. (30)

Bradford noted in his report (3) that the proximal segments of the limbs are more involved than the distal, differing in that respect from most other forms of polyneuritis. This observation has been confirmed by Pinckney (26), but others (8, 12) have observed predominant involvement of the distal segments.

Paresis of the trunk muscles is less common, although in fatal cases the disease has progressed upward to involve the intercostals and even the diaphragm.

The paralysis is of the lower motor neurone type. The muscles are flabby and toneless, although atrophy beyond that attributable to disuse is rare.

The deep reflexes are absent from an early stage, and this may be the only sign of motor involvement. The superficial reflexes are usually preserved.

A most common and significant symptom is involvement of the facial muscles. By some authors (35) facial paresis is regarded as an essential feature of the disease although it may also appear in alcoholic, diphtheritic and puerperal neuritides. In the cases reviewed, facial paralysis appeared in 35 out of 66 cases in which the sign was mentioned*, a percentage of 53. However, Holmes (12 cases) (12) states that facial involvement is "invariably present" and Bradford (30 cases) (3) that it is "almost always present". The involvement varies from a mere weakness of one side of the face to complete facial diplegia.

Other cranial nerves are less commonly involved. In 15 cases some evidence of cranial nerve involvement was mentioned.* Diplopia was seen in 4 cases, difficulty in swallowing in 6 cases, ophthalmoplegias in 4 cases, anisocoria in

1 case, deviation of the tongue in 2 cases, aphonia in 3 cases, nystagmus and palatal paralysis were each seen in one case.

The sphincters are sometimes involved, with incontinence of urine or feces. This was noted in 15 of 66 cases (23%).* Holmes (12) states that there is "almost constant" sphincter disturbance.

No fibrillary twitchings have been noted, nor any change in the electrical responses of the involved muscles.

Sensory Symptoms:

The sensory symptoms are very inconstant and not characteristic although usually present to some degree. Most frequent are paresthesias: numbness and tingling. Bradford (3) noted anesthesia and analgesia of the glove and stocking type which was relative rather than absolute. Proprioceptive sensations are often impaired in the absence of other sensory involvement. Strauss and Rabiner (31) noted segmental changes in sensory perception suggesting radicular involvement.

Laboratory Findings:

A moderate leucocytosis has been reproted in some cases (3, 15) although apparently it is not a common finding.

The spinal fluid changes are unique and when present are almost pathognomonic of the disease. This consists essentially of a greatly increased total protein without increase in cells. Guillain (11) in 1916 first described the finding

as an "albumino-cytologic dissociation". He regards it as an essential in the diagnosis of the disease, and states "Cases with a slight hyperalbuminosis with an albuminoid content of from 0.3 to 0.4 grams, do not belong to the syndrome or must be regarded as instances of an abortive form", It seems inadvisable to place such limits of variability for this sign without much larger series of cases than have been reported. He also remarks "...I refuse to recognize radiculoneuritis with hyperlymphocytosis or hypernucleosis as belonging to this syndrome". (10)

Of the 110 cases reviewed in this paper, * 40 were reported as showing an increased spinal fluid protein (above 40 mgm. per 100 cc.); 2 showed no increase; in 68 cases spinal taps were not reported. The amount of protein is usually above 100 mgm. per 100 cc. (29 cases), and often is enormous, in two cases (8) reaching 708 and 800 mgm. per 100 cc. respectively.

This albumino-cytologic dissociation, which persists in convalescence is a means of differentiating this relatively benign disease from poliomyelitis, encephalomyelitis and peripheral forms of epidemic encephalitis.

Albumino-cytologic dissociation may occur in poliomyelitis. The quantity of protein is, however, not so great and tends to fall at a time when a rise would occur in infectious neuronitis. (17) It is therefore important to examine

the cerebro-spinal fluid throughout the course of the illness and convalescence.

That a single spinal tap is not sufficient to make or rule out the diagnosis of infectious neuronitis is emphasized by Madigan and Marietta (15). They report one case in which eight spinal taps were done. The first, taken after the paralytic symptoms had become manifest showed only 18 mgm. per 100 cc. of total protein. But in the succeeding six weeks of the disease it rose to 122 mgm., the height of the curve being about 5 weeks after the onset. The curve gradually decreased then to normal.

A similar acellular hyperalbuminosis occurs in syphilitic polyneuritis, and occasionally in diphtheritic polyneuritis (11, 18). Merrit and Fremont-Smith (20) report it in 2 out of four cases of simple radiculitis, one case of lead polyneuritis, and one case associated with toxemia of pregnancy.

A word of caution is therefore necessary in regard to the diagnostic value of albumino-cytologic dissociation. Determinations must be repeated; clinical and etiologic evidence must not be disregarded. Until a large series of spinal fluid finding have been reported in other forms of polyneuritis, the finding of an increase in the total protein without pleocytosis, although highly suggestive of infectious neuronitis, can not be regarded as conclusive evidence of the disease.

Course:

The acute stage of the illness usually lasts 2 or 3 months, but convalescence is protracted. Occasionally the paralysis may be progressive, with eventual involvement of intercostals and diaphragm and death from respiratory paralysis. These cases were seen more commonly during the war, and the conditions under which they were observed may have contributed to their seriousness.

Gilpin, Moersch and Kernohan (8) report an average of 6.6 months for the process of recovery which was "practically complete and without recurrences". Bradford (3) remarked that two months was required for enough improvement to permit evacuation; six months for regaining complete health. Recovery is usually in the reverse order from that of the paralysis, the upper extremities recovering first, the legs last (3). In many the last to return are the tendon reflexes which may be absent for years after all other symptoms have disappeared. (10).

Prognosis:

In the 110 cases collected from the literature 22 fatalities occurred, a mortality of 20%. That this is not a true picture of the disease becomes apparent when it is realized that 10 deaths occurred in the 32 cases reported from the armies during the stress and strain of war conditions.

Of the remainder, 78 or 70% recovered more or less completely; in 10 cases the outcome was not reported.

Guillain (10) insists that the disease is essentially benign, recovery being the rule. The only residual he observed was persistent absence of the deep tendon reflexes, without causing symptoms. He maintains that "diseases with a high death rate are not the same as the Guillain-Barre Syndrome". (10)

Diagnosis:

Bradford (3) maintained that "acute febrile polyneuritis is a very definite clinical entity, capable of being separated clinically from other diseases of the nervous system." His criteria for diagnosis are: the constant bilateral affection of the face, the involvement of the muscles of the trunk, the progressive nature and occasional ascending character, the generalized muscular weakness rather than group paralysis, the involvement of proximal segments of limbs and the sparing of distal segments and the absence of obvious muscular atrophy.

Holmes (12) and Guillain (10) also believe that the disease displays symptoms characteristic enough to justify its place as a definite clinical entity, the latter exhibiting the spinal fluid findings as final proof.

The difficulties in diagnosis are well exemplified by the diverse nomenclature that has been employed in various reports. Each author believes that he has found the one characteristic symptom and includes that in the name which he gives to the disease; eg. "polyneuritis with facial diplegia" (30, 35)

"myeloradiculoneuritis (31), "radiculoneuritis with albumino-cytologic dissociation" (10, 11).

Taylor and McDonald (30) have especially emphasized the tendency of authors to magnify similarities and to minimize differences in symptomatology in the attempt to establish circumscribed clinical entities. They conclude that the "only possible generalization is that this infection of the nervous system is characterized mainly by widespread muscular weakness, lost deep reflexes and distrubed sensation of the neuritic or neuronitic type; together with a rather selective paresis of the facial nerves. It is impossible to bring the symptomatology into a definitely fixed type."

The faithfulness with which anatomical involvement of the nervous system is reflected in physical symptoms and signs is perhaps responsible for the present day confusion in neurologic nosology. We are in a period of transition from a classification on the basis of physical signs to a classification on the basis of etiology. It is obvious that the destruction or impairment of function of a given group of neurones, although producing an identical symptom complex in every case, may be of the most diverse etiology. Conversely a given etiologic agent may, depending on the structures affected, produce a bewildering array of clinical syndromes.

However, different etiologic agents seem to have affinities for different portions of the body, and their effects may be discernible quantitatively. In neuritis due to lead the palsy almost always picks out at first certain groups of muscles, such as the extensors of the wrists, and if it becomes general it is usually associated with that group of cerebral symptoms which have been described as lead encephalopathy. Arsenical neuritis is easily recognizable by the prominence of gastro-intestinal symptoms, pigmentation and trophic disturbances, serious sensory loss, early muscular atrophy, and its very slow convalescence; nueritis due to vitamin B deficiency is usually characterized by more pronounced and prolonged sensory symptoms and often accompanied by general symptoms such as edema, and vasomotor and cardiac aymptoms. Anterior poliomyelitis is characterized by a more acute onset with rapidly developing massive paralyses which do not progress. The fever at the onset is higher, and there is a leucocytosis in the blood and spinal fluid.

ETIOLOGY

Osler (24) in his original description of the disease stated that the onset resembled that of an acute infectious disease. Cobb and Coggeshall (6) offer the following useful classification of the neuritides:

I Localized
  a. Mechanical
  b. Infectious

II Generalized
  a. Virus
     Measles, small pox, chicken pox, herpes, acute infectious, poliomyelitis, encephalitis, rabies
  b. Bacteriotoxic
  c. Deficiency or metabolic
  d. Chemical

This classification includes infectious neuronitis with the virus group since the "clinical picture, the epidemiology, the fact that patients with encephalitis sometimes have a similar neuritis, and the few autopsies which show acute changes in the white and gray matter of the central nervous system are pieces of evidence pointing to a tentative classification of this with the known virus diseases."

Suggestive of an infectious etiology is the frequency with which a definite history of illness with general symptoms preceeding the onset of the paralysis is obtained. In 64 of the cases collected such a history was obtained.

The report of Bradford, Bashford and Wilson (3) contains the earliest and the only direct evidence of an etiologic nature. A study of the pathology of six fatal cases suggested a "septicemia or systemic poisoning which enters the central nervous system by way of the nerve trunks, both motor and sensory and probably of an infective nature." An emulsion from the spinal cord of fatal cases was injected subdurally into monkeys with rpoduction of typical symptoms and characteristic pathologic lesions. The disease was similarly transmitted from monkey to monkey. Wilson using Noguchi's anoerobic serum-

agar serum-broth tissue medium cultured tissue from two fatal cases and from four monkeys. He isolated from all six a minute coccoid organism arranged in pairs or short chains, gram positive in young culture, gram negative in older ones, with a deeply stained eccentrically placed portion and a narrow faintly stained margin. This organism "inoculated subdurally into a monkey reproduced the disease clinically and pathologically and finally the organism has been recovered post-mortem from the nervous tissue of the animal so inoculated."

It would thus appear that Koch's postulates had been fulfilled in regard to this disease. Although the work has never confirmed, there are no reports in the literature of similar experiments, aimed at either confirmation or disproval. The etiologic significance of the organisms has never been widely accepted and the general opinion held at present is that the disease belongs with those caused by a virus.

The similarity of this disease to poliomyelitis and epidemic encephalitis, both of virus etiology, has suggested to some investigators that a virus may be the responsible agent here. Foster Kennedy in discussing cases of myeloradiculoneuritis presented by Strauss and Rabiner (31) compared them with cases seen in an epidemic in Massachusetts. In a hospital for mental patients there was an epidemic, traceable to streptococcus infection in milk, in which many died and many more were affected with polyneuritis, radiculitis, and myelitis.

Barker (1) introduced the term "septineuritis" to describe the process by which a virus injected into the brain may become generalized throughout the whole central nervous system and is recoverable later not only from all parts of the central nervous system but from the peripheral nerves and sensory and sympathetic ganglia. Similarly if virus is injected into the sciatic nerve it will cause the same change in the spinal cord, nerve roots and root ganglia that follow injection into the brain. He suggested that infectious neuronitis was a septineuritis due to a Schwannophil virus.

Gilpin (8) M ersch and Kernohan concluded from their clinicopathologic studies of 20 cases that they were dealing with a condition due to a virus that in the present instance has a predeliction for the peripheral neurones.

The recent work on the elucidation of the role of dietary deficiencies in the production of polyneuritis has thrown a shadow of doubt over the toxic and infectious etiology. Levy* has expressed the belief that all forms of polyneuritis have a common origin in nutritional deficiency.

PATHOLOGY

There are few references in standard works on pathology to the anatomic changes which take place in polyneuritis. This may be due to the infrequency of fatalities from this disease.

* Cited in Masten, Mabel (16)

Bradford, Bashford and Wilson (3) had the unique opportunity of examining tissues from six fatal cases of infectious polyneuritis. Grossly they found only edema of the brain and cord with congestion of the meningeal vessels, and some petechial hemorrhages on cross section. Microscopically the peripheral nerves showed Wallerian degeneration of recent origin with proliferation of the cells of the sheath of Schwann. These changes were more marked in the nerves of the lower than in those of the upper extremities, and more marked in the motor than the sensory nerves. Characteristically isolated nerve fibers or groups of fibers were affected leaving intervening ones free. The degenerative changes were accompanied by an acute neuritis with an enflammatory exudate of round cells and hemorrhages scattered throughout nerve bundles, with degeneration of the nyelin sheath and fragmentation of the axis cylinders.

The spinal cord showed degeneration of the cells of the anterior and posterior horns scattered throughout the cord with intervening normal cells. These degenerative changes were characterized by eccentricity of the nucleus, clumping of the tigroid substance and infiltration of round cells about such degenerated nerve cells. There were no perivascular collections of round cells. Neuronophayg was observed in some cases.

In one instance the lumbar and dorsal posterior root

ganglia showed eccentricity of nuclei, vacuolation of cytoplasm and clumpin or disappearance of tigroid substance. Between the cells was a patchy accumulation of round cells.

The brain showed little that was abnormal.

The authors summarize their findings thus: " the foregoing details...indicated clearly a general involvement of the grey matter of the entire nervous system with a gradually ascending progression, and are compatible with a neuritis dominating the early clinical features. There are clear indications that the nerve cells of the cord are involved early, and that the diffuse accumulation of round cells is a later phenomenon to which their aggregation around damaged or degenerated nerve cells succeeds at a still later stage. Consideration of the whole pathological process would point to a septicemia or systemic poisoning which enters the central nervous system by way of the nerve trunks, both motor and sensory, and probably of an infective nature."

Cassamajor (5) in discussing Bashfor's work on the pathology of the disease, states that "many of the changed cells that he describes and pictures, especially the shrunken ones with crenated outlines, appear more typical of post-mortem changes than any known type of cell degeneration. Besides the changes in the nerve cells, Bashford describes

a marked increase in the number of small round cells in the gray matter of the cord. It is evident that these cells are small, young neuroglia cells and not cells of inflammatory mesodermal origin."

Cassamajor presents the pathologic description of two cases of his own. These were characterized grossly by hyperemia, hemorrhage, edema with fibrous swelling in the arachnoid and thickening of the pia. Microscopically there were no changes in the blood vessel walls and no round cell infiltration. There was an increase in the cellular neuroglia in the central gray matter, around the root fibers and in the posterior root ganglia. There was marked degeneration of both primary and secondary character of the nerve fibers, most marked in the motor fibers and almost limited to where they run in the arachnoid just after leaving the pia. There was evidence of beginning degeneration in the anterior horn cells and some tract cells. There was marked degeneration of the posterior root ganglion cells with definite neuronophagy.

He summarizes his findings thus: "when the organisms reach the central nervous system they produce there two definite changes: 1. In the mesodermal elements of the arachnoid and pia the result is edema and hemorrhage, with some increase of connective tissue cells, without any of the usual signs of inflammation, i.e. infiltration with white blood cells.

2. The changes in the ectodermal elements appear to be most marked where the greatest mesodermal changes take place, i.e. in the arachnoid where the nerve roots run through it. The nerve fibers here show marked degeneration of a toxic and infectious nature. In the peripheral nerves the degeneration may be looked on as both secondary and toxic in nature. In the spinal cord and posterior root ganglions we see the usual picture of infectious toxic degeneration, with some evidence of early secondary degeneration due to the destruction of the fibers in the arachnoid. The increase of the neuroglia cells, the degeneration of the nerve cells and the neuronophagy are all parts of the infectious-toxic picture.

"The question of the proper name for the disease is still an open one. To call it an acute arachnoiditis appears very inadequate, and hence the writer has been obliged to fall back on the clinical manifestations of the disease for the title of this paper. Perhaps the term "Acute infective meningomyeloneuritis" might be acceptable."

Holmes (12) also observed degeneration in the peripheral nerves and changes in the ventral horn cells. Gilpin, Moersch and Kernohan (8) reported pathologic studies on three cases. They found that the degeneration was limited to the peripheral nerves, without changes in the cord. The changes in the peripheral nerves correspond closely with those described above.

It is quite obvious from the pathologic descriptions

given that this is a true disease of the nervous system. The process is diffuse, affecting both central and peripheral nervous systems, although the peripheral system is more severely damaged than is the central.

SUMMARY

The literature concerning a rather rare and obscure disease of the nervous system has been reviewed and the present knowledge of it summarized. It is characterized clinically by an onset resembling that of an acute infectious disease, a progressive paralysis of a neuritic type with sensory symptoms of a radiculitis, and by a peculiarly frequent and isolated involvement of the facial nerves. The characteristic laboratory finding is of a very high spinal fluid protein without an associated pleocytosis. The etiology is obscure, although its relation to the virus diseases has been postulated. Pathologically there is a widespread toxic-infectious involvement of both peripheral and central nervous systems.

The proper name for the disease is in dispute and the one used as a title for this paper is tentatively selected because it seems to come closest to describing the pathologic process simply. A summary of the clinical findings in 110 reported cases is presented.

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