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PATHOLOGIC CHANGES IN THE OPTIC SYSTEM IN DISSEMINATED SCLEROSIS

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From the literature pertaining to disseminated sclerosis, and from my own clinicopathological observations (1952, 1953), I cannot but conclude *a)* that there is probably more than one factor playing a part in the pathogenesis of the disease, and *b)* that it is always another one of them that exerts its effect in the different stages of the disease.

I distinguish three stages in disseminated sclerosis, viz. — (i) the stage of onset; (ii) the latent period; (iii) the developed or demyelination stage.

By the fact that in a certain proportion of cases in the initial stage retrobulbar neuritis occurs, attention has been drawn to the optic system.

In retrobulbar neuritis, there is little precise knowledge as yet as to what is really taking place in the optic nerve, but the passing character of the ophthalmic disease and the course of disseminated sclerosis make it appear probable that minute foci surrounded by oedematous zones develop. Knowing the inflammatory oedematous character of »fresh foci« in disseminated sclerosis, it is assumed that similar foci may arise in the optic nerve.

This assumption appears to be confirmed by the changes of the optic system in autopsied, i. e. fully developed, cases of disseminated sclerosis.

Already *Oppenheim* (1887) described in five autopsied cases demyelinated foci in the optic nerve, the optic tract, and the chiasma, and emphasized the possibility of their presence even when vision is unimpaired and the ophthalmoscopic finding negative. In 11 out of his 20 cases, i. e. in more than 50 per cent, of them, he observed pathologic changes in the ocular fundi and alterations in the visual field.

Uhthoff (1890) found in five of his six autopsied cases pathologic changes such as sclerotic foci, simple atrophy, and »interstitial neuritis« in the optic nerve and the chiasma. The small vessels and capillaries were dilated and replete with blood, proliferation of the cellular elements in the vessel walls was marked, and inflammatory reaction was present around the larger vessels and in the distended connective tissue septa.

Völsch (1908) found one small focus of disseminated sclerosis in the chiasma, several foci in the optic nerve and tract, and a single small one in the right pulvinar of the thalamus.

Flatau and Kölichen (1911) encountered in two out of four cases foci in the optic nerve ; in the one they observed complete absence of myelin, sclerosis and thickening of the pia-arachnoidea ; in the other, marked demyelination and thickening of the vein wall.

Velter (1911, 1912) described foci in the optic nerve and tract, in the chiasma, and the optic radiation.

Rönne and Wimmer (1913) noticed in the chiasma a very large sharp-edged plaque infiltrating the optic tract, and in the optic nerve they observed disseminated degeneration and almost complete degeneration of the ganglionic cellular layer in the left bulbus.

Siemerling and Raecke (1914) encountered in the optic nerve in eight cases atrophy of various degrees, glial sclerosis, and, in one case each, round cell and plasma cell infiltration respectively.

Taylor (1922) found in three cases sclerotic foci or fully developed sclerosis in the chiasma, the optic nerve and the tract.

Lisch (1933) observed in 12 cases of disseminated sclerosis marked plaques, diffuse medullary lesions, perivascular lymphocytic infiltration, in some instances also »rarified perivascular foci«, in the chiasma, the optic nerve and tract, as well as (*Lisch* and *Hermann*) in the lateral geniculate body.

The latest books on neurohistopathology *(Jakob, Biggart, Lichtenstein)* and the more important contributions to the subject, all emphasize the presence of foci in the areas mentioned. In this connection, *Marburg*'s figures Nos. 7 and 249, *Pette*'s figures Nos. 105 and 151, *Zimmerman* and *Netsky*'s No. 109, and *Adams* and *Kubik*'s No. 8, merit particular attention.

In my opinion, the rest of the visual pathways shows also a specific disposition to participate in the process. According to the clinical observations of *Savitsky* and *Rangell*, changes in the optic radiation occur in 46 per cent of the cases, a fact which *Zimmerman* and *Netsky* correlate with »the very frequent lesions« of the white matter around the posterior horn.

With a view to checking the correctness of my assumption, I endeavoured to control in twenty autopsied cases of disseminated sclerosis the above data referring to lesions in the central and peripheral visual pathways.

The following Table shows that *proper foci of disseminated sclerosis* were most frequently encountered in the white matter around the posterior and the lower horns, in the chiasma, and the optic tract and nerve.

The column »other lesions« is meant to indicate that, whereas there were no foci of disseminated sclerosis in the chiasma and the optic nerve or tract, respectively in two cases, in the lateral geniculate body in ten cases, and in the thalamus and the white matter around the lower horn in one case each, *there were, on the other hand, histological changes to be observed which represent features that are essential from a general pathological point of view.* Such features are : dilatation of capillaries and small vessels, precipitation of fibrin in the perivas-

Pathologic changes in the optic system of 20 autopsied cases

Site of lesion	Number (and percentage) of cases showing		Total number of cases
	foci of disseminated sclerosis	other lesions	
Chiasma	11 cases 55 per cent	2 cases	13
Optic nerve or tract	9 « 45 « «	2 «	11
Lateral geniculate body	5 « 25 « «	10 «	15
White matter around posterior horn	17 « 85 « «	—	17
White matter around lower horn	10 « 50 « «	1 case	11
Thalamus	6 « 30 « «	1 «	7

cular space, in some instances more or less marked infiltration by lymphocytes (Fig. 1, lateral geniculate body), hyaline changes in the vascular walls, an increased number of adventitial fibres filling the dilated perivascular space, sometimes almost completely (Fig. 2, chiasma), foci of »spongy« appearance of the nervous tissue (Fig. 3, optic nerve), proliferation of interfascicular connective tissue (Fig. 5, optic nerve). Very interesting are the secondary changes in the geniculate body in cases Nos. VI, VII, XI, and XII : here the medullary fibres penetrating from the optic tract are spongy, distended, and disintegrated, and the nervous cells show fatty degeneration.

The thalamus has been included in the Table because there exist internuclear connections between the lateral geniculate body on the one hand, and the pulvinar of the thalamus, respectively the ventral and lateral thalamic nuclei, on the other hand ; besides, because it is known from the latest investigations by *Hartmann and Simma* that areas 18 and 19 (lateral occipital gyri) are in connection with the pulvinar nucleus.

If the essential histological features now unders tood to be included under the heading »other lesions« were added to the percenta ges shown in the above Table we would be justified in stating that foci of disseminated sclerosis or different other pathological lesions have been found :

in the chiasma in 65 per cent of the cases

in the optic nerve or tract in 55 per cent of the cases

in the lateral geniculate body in 75 per cent of the cases

in the white matter around the posterior horn in 85 per cent of the cases (unchanged)

in the white matter around the lower horn in 55 per cent of the cases

in the thalamus in 35 per cent of the cases — under review.

Let us now proceed to the photographs of some of our illustrative cases .

Foci in the *lateral geniculat e body* and the *semioval center* can be seen in the Weigert preparation of case No. XVIII (Fig. 4). Foci in the *chiasma* and the *optic nerve* are shown in sections stained according to Spielmeyer (Fig. 6—11).

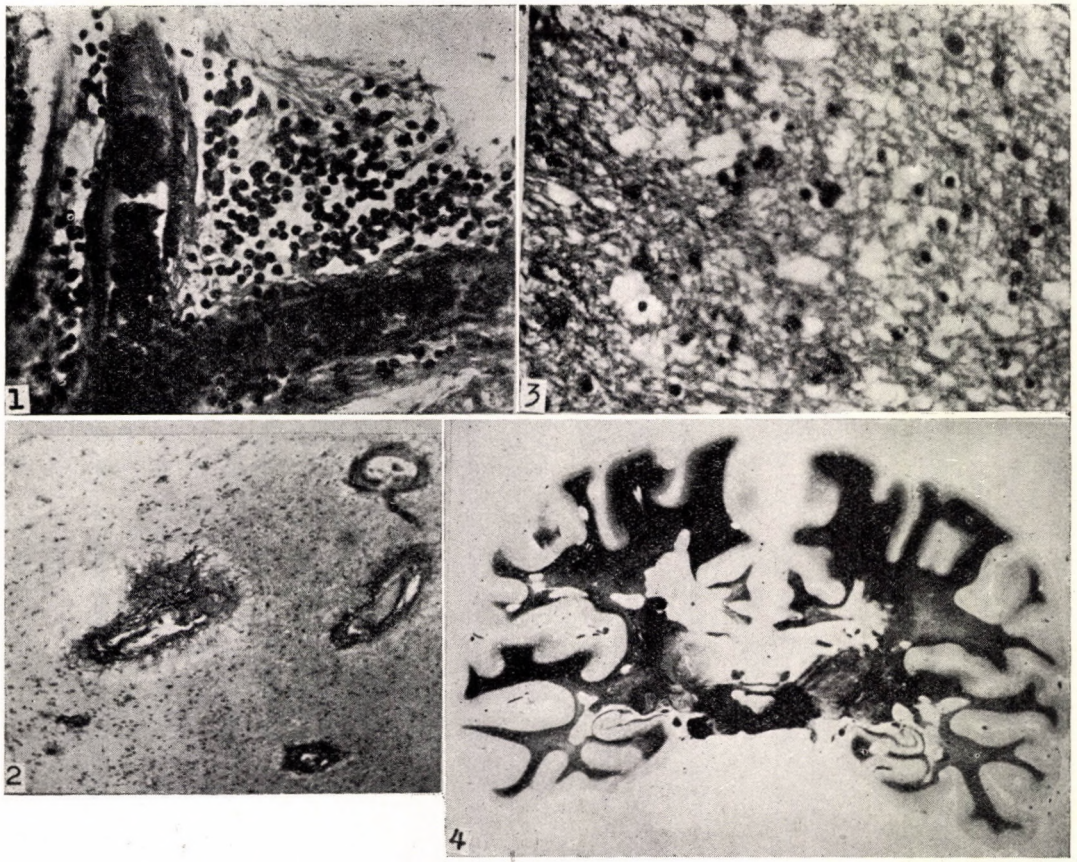

Fig. 1. Case No. XIV. Lateral geniculate body. Haematoxylin — van Gieson. Many lymphocytes in the dilated perivascular space with fibrin threads amongst them. *Fig. 2. Case No. XI.* Chiasma. Haematoxylin — van Gieson. Vessel walls thickened and fibrillary; dilated perivascular space filled with adventitious fibres. *Fig. 3. Case No. XII. Optic nerve.* Haematoxylin — van Gieson. »Spongy« area in no communication with vessel. *Fig. 4.* see in text.

Fig. 9 and 10 presents a micro-focus in the optic nerve. Fig. 11 shows fat-negative degenerative stripes between intact myelin-bundles in the optic nerve, with marked destruction and fragmentation of myelin. Fig. 12 demonstrates a case where, although the focus in the optic nerve is of old standing, the pial membrane contains cells with numerous fatty granules, indicating that the process cannot yet be regarded as completed. The focus of the *lateral angle* and that of the *lower horn* are shown in a myelin- sheath preparation (Weigert--Pál) in Fig. 4.

The photograph of case No. IX (Fig. 13) represents a characteristic picture of the foci around the *lower horn*. Foci in the white matter around the lower horn can be observed in Fig. 14. In Fig. 15 the eye is caught by subcortical demyelination in the region of the fissura calcarina, partially sparing the »U« fibres ; under low power magnification this appears in case No. V. as a characteristic »undulating« structure. (Fig. 16).

Marburg, and later many other authors have described »maplike« scattered or concentric foci, besides which foci characteristic of disseminated sclerosis were also observable (*Hallervorden* and *Spatz*, *Juba*, *Hechst*, *Steiner*, *Sträussler and Scheinker*, *Benoit*, *Peters*, *Ferraro* and *Jervis*, *Pette*, etc.). In between, in 1927 and 1928, *Baló* has published his case of »encephalitis periaxialis concentrica«, which gave rise to much scientific interest. *Baló* later re-named this disease »leukoencephalopathia concentrica« and insisted upon its being independent of both the disseminated and the *Schilder*-type diffuse sclerosis. Some of the research workers, with *Barré and van Bogaert* in the lead, have accepted this standpoint, while others, chiefly *Hallervorden* and *Spatz*, *Ferraro*, etc., refuted it, declaring the concentrated focus to be a specific histological *detail-structure* ; all the more so as similar histological change had been observed in *Schilder*-type diffuse sclerosis by a long chain of investigators (*Barré*, *Morin*, *Draganesco* and *Reys*, *Waggoner* and *Löwenberg*, *Zeman*, *Ule* etc.). Since similar observations had been made by *Ferraro* and *Jervis* in acute encephalomyelitis and in potassium cyanide poisoning, and by *Lehoczky* in chronic CO-poisoning, it seems to be obvious that concentric sclerosis is a special form of degeneration of the white matter, which throws light on the genesis of the pathological processes in that area. According to *Lhermitte*, it is quite possible to reconcile the »myelolytic diffusion theory«, which is based upon the structure of the Liesegang rings (*Hallervorden* and *Spatz*), with the allergic concept. In such cases the antigen-antibody conflict would run its course on the model of the said rings.

In our opinion, the fact that concentric demyelination and its preceding stage, respectively, appear in different clinical pictures is indicative of a common pathomechanism in the leukoencephalitides. This should, of course, not be resorted to for the purpose of drawing any conclusions in respect of aetiology.

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In summing up the above data of the literature and our own histological findings we feel bound to state that since in disseminated sclerosis lesions in the extra-cerebral as well as in the intra-cerebral parts of the optic system appear to be of striking frequency, the optic system ought to be regarded as one of the sites predilected by the disease

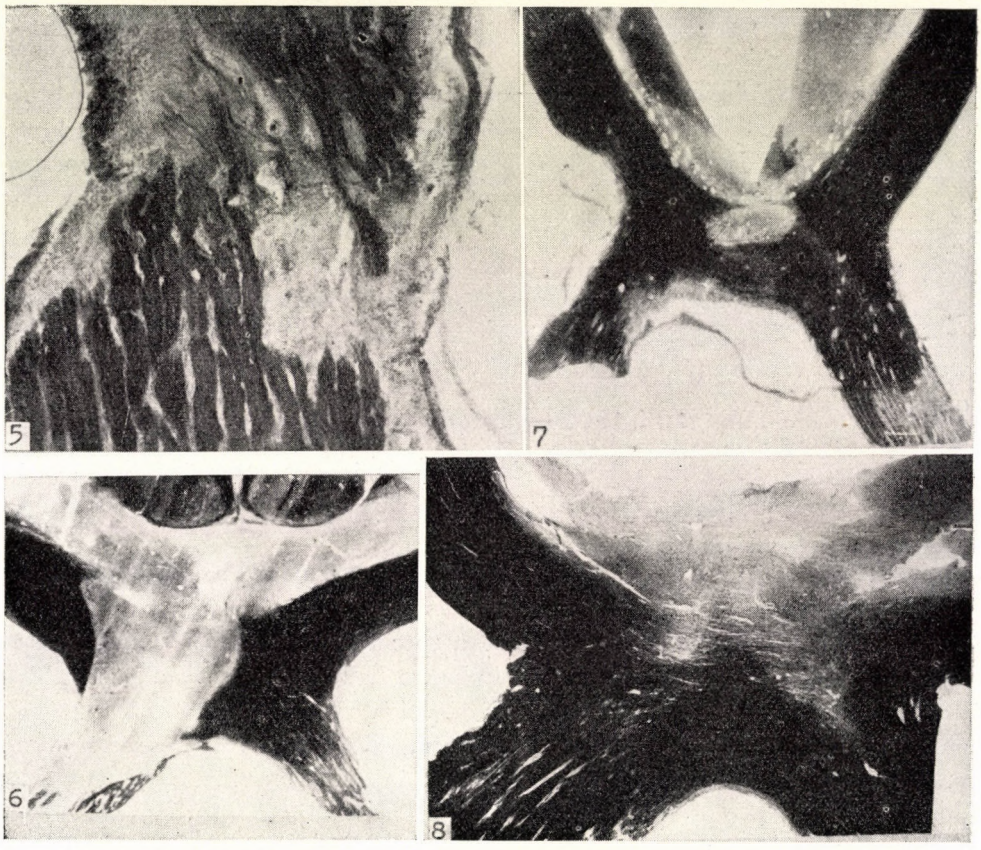

Fig. 5. Case No. I. Optic nerve. Herxheimer's scarlet-haematoxylin. Scar tissue with smal foci amongst bundles of nerve fibres.
Fig. 6—8, see in text.

Wishing to somewhat expound this statement I would refer to a working hypothesis brought forward by me and published in some previous papers, the essential points of which are the following.

As already mentioned, three stages are to be distinguished in disseminated sclerosis : the stage of onset, the period of latency and the demyelination stage.

In the initial stage, or stage of onset, an unknown infectious agent, *which may be a virus*, penetrates the organism from the external environment (*McAlpine*; *Margulis*, *Soloviev* and *Shubladze*).

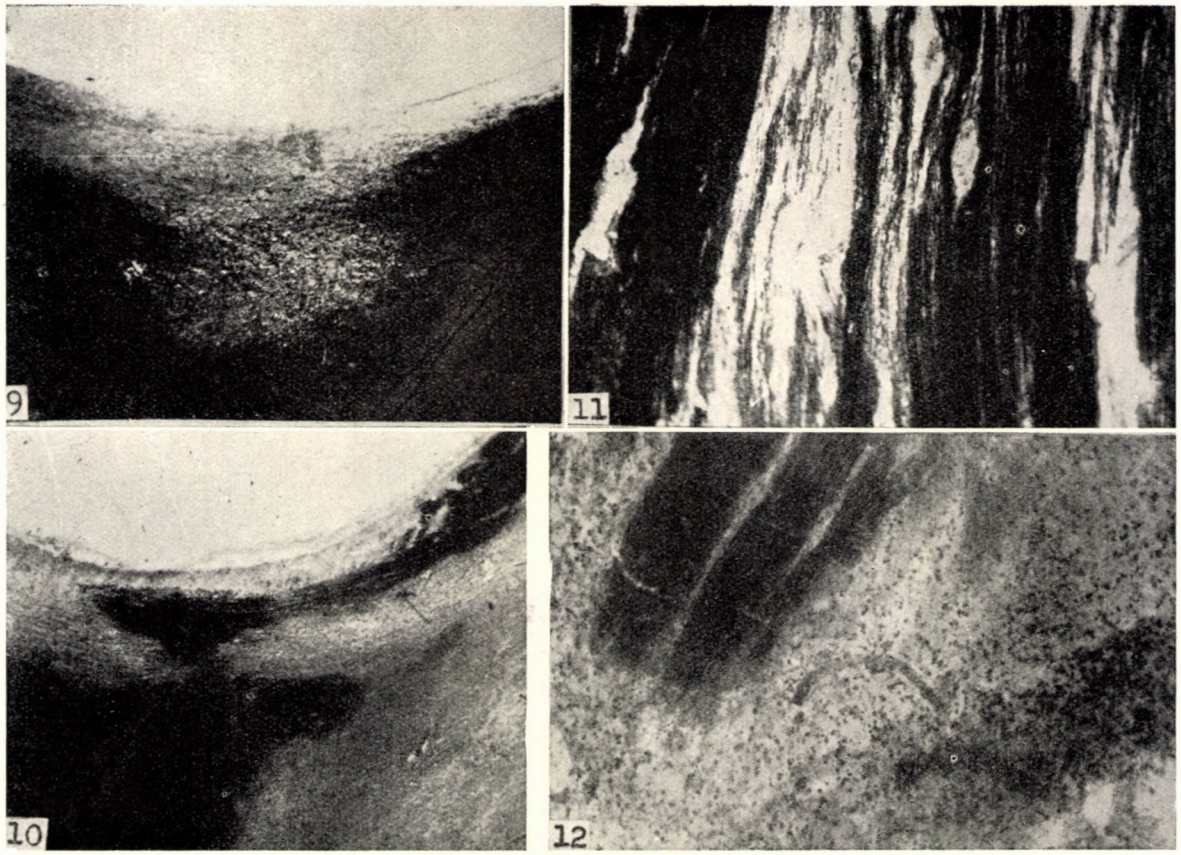

Fig. 9—11, see in text.

Fig. 12. Case No. I. Optic nerve, Herxheimer's scarlet-haematoxylin. Many fatty granular cells in the pia mater.

According to some authors *(McAlpine)*, the skin, the subcutaneous tissue, the throat and the intestinal tract can be sites of penetration. A possible addition to these, which merits serious consideration, is *the eye*. This seems to be corroborated by all the clinical data reporting that in a high proportion of

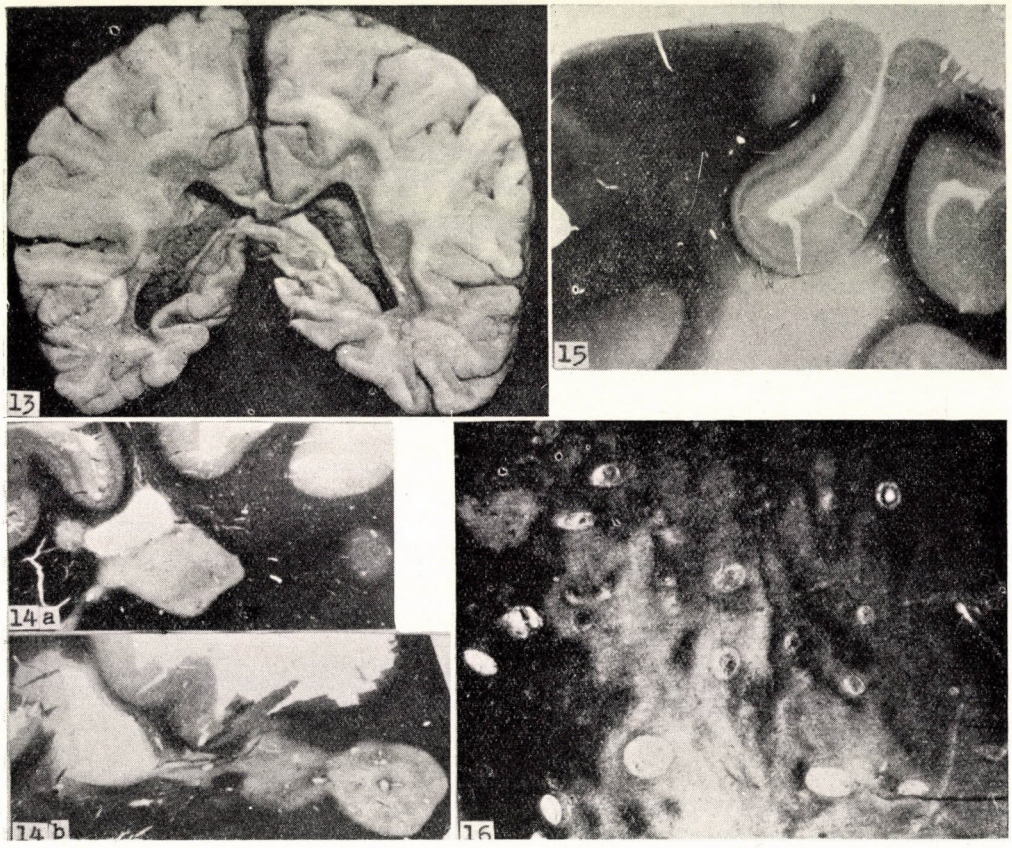

Fig. 13. Case No. IX. Vast sclerotic foci around the central part and the lower horn of the lateral ventricles.

Fig 14, see in text (Spielmeyer's myelin stain).

Fig. 15. Case No. IX. Occipital lobe. Spielmeyer's myelin stain. Subcortically situated demyalinated focus.

Fig. 16. Case No. V. White matter of occipital lobe. Herxheimer's scarlet-haematoxylin. Degenerated areas showing particular undulating, »map-like« tracery. Fatty granular cells in the dilated perivascular spaces.

the patients retrobulbar neutritis precedes by several years, even decades, the clinical syndrome of disseminated sclerosis (further details on this point follow below).

I believe that it is by drop infection that the infectious agent first finds its way to the conjunctiva, from where it drifts into the limbus to be passed

through the communicating veins into the ophthalmic vein, then to the optic nerve, from there to the chiasma, reaching finally the optic tract.

It has been definitely established that some of the known virus diseases can be transmitted to the conjunctiva. Such virus diseases are rabies, poliomyelitis, lethargic encephalitis, Japanese encephalitis type B, Eastern encephalomyelitis, acute lymphocytic choriomeningitis, ascending myelitis, chickenpox, and inguinal lymphogranuloma. In some of these spontaneous infection is known to have occurred via the conjunctiva; moreover, in a few instances accidental corneal infections are reported to have happened *(Lépine and Sautter)*.

Since in disseminated sclerosis retrobulbar neuritis is a transient phenomenon and the patient's vision is restored after the lapse of a few weeks or days, or sometimes even within a few hours, it seems to be evident that the foci causing the optic neutritis are small ones with a tendency to rapid regression. From a pathological point of view they are in all probability reversible, inflammatory, oedematous foci which only turn into irreparable, scarred foci if the process is repeated *(Foster Kennedy)*.

Since the observations of *Oppenheim* (mentioned in the foregoing), autopsied cases have been reported by *Uhthoff*, *Wilbrand* and *Sänger*, *Hermann*, *Marchesani*; in which, in spite of the presence of very marked optic foci the ocular fundi were found to be completely normal (see *Uhthoff*'s case No. IV, and *Marchesani*'s Figs. Nos. 19, 20, and 21).

The explanation of this contradiction may be that in these cases the visual upset is caused less by the foci than by the inflammatory oedema accompanying them.

Theoretical importance attaches in our opinion to the case reported by *Oppenheim* (1914), in which the onset of disseminated sclerosis manifested itself in the optic nerve of a 34 years old female patient and for 10 to 15 years always »returned« to this site and remained confined to it, which really means that the optic system alone fell ill. *Oppenheim* offered the explanation that the area once injured remained a »locus minoris restistentiae«. These were the cases which he called the *optic or ocular form* of disseminated sclerosis.

How is the recurrence of the initial symptoms taking place? In my opinion it is not due to any additional infective agents finding their way into the organism but to the fact that the formation of the small primary foci in the optic system involves decomposition of myelin sheaths, and that the decomposition products induce the production of an antibody in the nervous tissue of the opticus.

The antibody or antigen is carried by the blood to every part of the organism and thus also to the central nervous system, and if in the later (third) stage some external adverse effect, such as a cold, pregnancy, emotional or physical strain, happens to befall the organism, there arises in the sensibilized nervous tissue an initially small but continuously growing demyelinated area, the focus of disseminated sclerosis.

This, then, were the explanation of the possible appearance of myelinolytic foci in the later stage of the disease at any place where there are myelin sheaths, i. e. in any part of the central nervous system, moreover in the cerebral and peripheral nerves.

What has been set forth in the foregoing is considered a working hypothesis, and there are several side-issues attaching to it.

There is, for instance, the problem presented by the *periventricular and subependymal location* of the foci in disseminated sclerosis, which caught the attention of even the earliest investigators. At one time this was thought to be explainable by the proximity of the cerebrospinal fluid and, respectively, by the presence of a supposed pathologic agent in it. However, the perivascular origin of the foci was pointed out by *Pette* as early as in 1929. (See Fig. No. 157 in his book: »Die akut entzündlichen Erkrankungen des Nervensystems«). *Döring*, in 1939, observed smaller foci around the posterior horn separated from the surface of the ventricle by a stripe of intact myelin (see his Fig. 3). I have made similar observations and therefore share *Döring*'s opinion that the periventricular, continuous, large focus should not be utilised in any pathomechanistic or pathogenetic essays. But apart from this, the frequency of periventricular focus formation remains an open question to which there is as yet no definite answer. It might be a peculiarity of the vascular system *(Schob)*, but it might equally be that several other factors are playing a part in it. Amongst these factors, in my opinion, significance attaches at the above-enumerated areas of the ventricular system — to the optic system as a predilected site. The frequency of lesions in the white matter around both the posterior and lower horns is made comprehensible by the *visual radiation of Gratiolet* proceeding from the lateral geniculate body towards the occipital cortex (see Table I).

Another problem is, why are there often lesions to be found in the white matter around the posterior horn in different other demyelinating processes? Thus, e. g. in disseminated encephalomyelitis, in which, according to *Környey*, perivascular lesions occur frequently in the white matter of the occipital lobe. (See Fig. No. 13 in his monograph: »Myelitis«.)

Környei, and also *Döring*, deem it a reasonable answer to this question that the two pathologic forms are closely related and that the common basic disturbance (»Grundstörung«) is to be sought for in the hyperergic mechanism. It seems, however, that aetiopathogenic research alone will only be capable of supplying a reassuring solution and, in my opinion, the problem should be kept in suspense until clarification of the aetiology will have been achieved.

The clinical symptoms of the optic system and their significance in disseminated sclerosis have long since occupied the minds of research workers.

It is known that in *retrobulbar neuritis* the visual upset (amblyopia, »hazy« vision) begins with relative colour-scotoma, which later turns into absolute colour-scotoms, and then into absolute scotoma. As an after-effect of retrobulbar

neuritis and papillitis respectively, the temporal half of the papillae, or on rare occasions the entire papilla, grows pale, yet the disease leads very seldom to complete blindness: in 1,8 per cent of the cases *(H. D. McIntyre* and *A. P. McIntyre)* The probable reason of this is that the axones are spared for a long time.

The literature on the question of the frequency and significance of retrobulbar neuritis in disseminated sclerosis is imposing. We refer to the comprehensive works of *Kyreleis*, *Marburg*, and *Marchesani*.

The views differ greatly; one of the most important causes of the differences in opinion is perhaps the fact that in their studies the authors depart now from retrobulbar neuritis, now from disseminated sclerosis.

Thus, while *Gunn*, the ophthalmologist, observed disseminated sclerosis in 22,8 per cent of 223 cases of retrobulbar neuritis, *Behr* in *Siemerling*'s Neurological Clinic diagnosed retrobulbar neuritis in 75,4 per cent of the patients suffering from disseminated sclerosis and stated that in one third of the cases the former condition manifested itself as an early symptom. *Marburg*, the neurologist, diagnosed disseminated sclerosis in 55 per cent of his retrobulbar neuritis cases. The ophthalmologists *Bruns* and *Stölting* observed lesions in the optic nerve in 58 per cent of their cases of disseminated sclerosis, and in 32 per cent of them isolated visual upset in the *early* stage of the disease. The ophthalmologist *Windmüller* found changes in the papilla in 57 per cent of 90 patients with disseminated sclerosis in the Strümpell Clinic, but retrobulbar neuritis as an initial symptom occurred in 46 per cent only.

Yaskin, *Spaeth* and *Vernlund* diagnosed retrobulbar neuritis in 55 per cent of multiple sclerosis, but only in 25 per cent as an initial symptom. In *Ragnar Müller*'s 793 patients the corresponding figures were 54 and 20 per cent, respectively.

All these figures represent the more frequent »mean values«. Of the extremes I would point out the 70 per cent of each *Curschmann* and *Kampherstein*, and *Adie*'s 73,1 *Behr's* 75,4 per cent, also the 22 per cent of *Leinfelder* and the 13,9 per cent of *Adams* et *al.*

The following may be further reasons for this divergency: 1. dissimilarity of the material examined; 2. the specific province of the investigator (whether ophthalmologist or neurologist); 3. the patient's ignorance of the interdependence of symptoms, owing to which they do not observe them and are, therefore, unable to supply adequate information; 4. an overlooking of the fact that retrobulbar neuritis will not always develop into disseminated sclerosis, the same as only in a certain proportion of the cases will primary chancre develop into neurosyphilis; 5. the failure of authors to compile their statistical data on a uniform basis. It is obvious that those who report *pathologic changes in the optic nerve* in general, as for instance *Carter*, *Sciarra* and *Merritt** (78 per cent), must arrive at values different from those contained in statistical returns concerned solely with retrobulbar neuritis.

In a *smaller proportion* of the cases retrobulbar neuritis is followed as early as within a week or two by the other symptoms of disseminated sclerosis, but in three quarters of them the characteristic syndrome will not appear until after a few years, or even decades.

Already *Oppenheim* (1887) pointed to the *period of latency* or »free interval« embracing possibly tens of years. These have been confirmed by *Strümpell*, *Fleischer*, *Windmüller*, and others. In *Oloff*'s review (1917) of the pertaining literature the period of latency is shown to extend from 8 to 46 years. According to my own observations it lasts from 2 to 15 years.

Elucidation and significance of the latency period are subjects outside the scope of the present publication.

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In conclusion, it needs to be mentioned that the 20 cases on which this paper is based all belong to the chronic form of disseminated sclerosis. Of them, 16 were slowly progressive cases, 2 progressed in »outbursts«, and 2 belonged at the beginning to the first, later to the second group.

As regards *extension*, the *cerebrum* participated in the pathologic process in all the 20 cases, in 17 of them in a very marked manner, in 3 to a lesser degree. In the *spinal cord*, foci were found in 18 cases, *in 2 they were completely absent*, in one of the latter the disease lasted for 4, in the other for 8 years. *Hechst (Horányi)* in an acute case, and *Adams and Kubik** in a chronic one lasting for 23 years, also found the spinal cord to be completely normal. In the earlier literature a purely cerebral localisation had been described by *Fürstner*. In the *cerebellum* we encountered foci in 7, and in the *medulla oblongata* and the *pons*, respectively, in 14 cases.

In the clinical picture it is practically always the spinal symptoms that come into prominence (spastic paraplegia, incontinence, etc.). This is probably because within the narrow spinal cord even small foci will cause conspicuous symptoms. On the other hand the foci in the central white matter, and the sometimes very extensive ones around the posterior and lower horns, etc., may not give rise to clinical symptoms at all.

Summary

Twenty cases of disseminated sclerosis have been examined histologically. Characteristic foci have been observed in the chiasma in 55 per cent, in the optic nerve or tract in 45 per cent, in the lateral geniculata body in 25 per cent, in the white matter around the posterior horn in 85 per cent, in that around the lower horn in 50 per cent, and in the thalamus in 30 per cent of these cases.

In some of the negative cases lesions have been encountered in the above enumerated sites which from pathological point of view are considered to be of significance: dilated capillaries, stasis, hyaline vessel walls, rarefication of tissues, dilatation of the perivascular space and serous precipitation, fatty degeneration of nerve cells, degeneration of myelin sheaths.

On the basis of our data we regard both the extra- and the intra-cerebral parts of the optic system as sites predilected in the process of disseminated sclerosis

Our pathological data appear to be in conformity with former as well as with most recent clinical observations

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b) *Contributions :*

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ПАТОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ ЗРИТЕЛЬНОЙ СИСТЕМЫ ПРИ РАССЕЯННОМ СКЛЕРОЗЕ

Т. Лехоцки

Резюме

Автор наблюдал 20 случаев рассеянного склероза и при гистологических исследованиях он нашел характерный очаг в перекресте в 55%, в зрительном тракте или нерве в 45%, в боковом коленчатом теле в 25%, в белом веществе около заднего рога в 85%, в белом веществе около нижнего рога в 50% и в таламусе в 30% всех случаев.

Кроме того в одной части отрицательных случаев он наблюдал на вышеприведенных местах изменения (расширение капиллярных сосудов, стаз, гиалиноз стенки кровеносных сосудов, поредение ткани, расширение периваскулярной полости и серозное

выпадение, ожирение нервных клеток, дегенерация миэлиновой оболочки), которые он считает весьма значительными с общей патологической точки зрения.

На основании своих данных, автор считает как внецеребральную, так и внутрицеребральную часть зрительной системы предилекционным местом в процессе рассеянного склероза.

Патологические данные автора совпадают как с прежними так и с новейшими клиническими наблюдениями.