and operative technique, and with the limitation of the radical operation to cases accurately localized.

4. The palliative operation is strongly indicated to relieve symptoms where localization cannot be accurately made or the tumor cannot be removed. The exploratory operation is contraindicated.

5. Practically all circumscribed growths of moderate size are suitable for operation.

6. The osteoplastic method should be employed and the most rapid and perfect technique adopted which the circumstances allow.

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MULTIPLE SCLEROSIS WITH DEMENTIA: A CONTRIBUTION TO THE COMBINATION FORM OF MULTIPLE SCLEROSIS AND DEMENTIA PARALYTICA.*

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INTRODUCTION. Disseminated sclerosis of the central nervous system is generally accompanied by slight mental changes. In the larger number of cases the psychical alterations are insignificant, of mild character, and playing a minor rôle in the list of symptoms. They may be summed up briefly as follows: mental dulness and apathy; mild states of exaltation or depression and emotional instability; undue mental complacency and contentment.

With the progression of the affection and the increase of the general symptoms the mental impairment suffers still more, so that in the later stages a partial dementia is not uncommon.

Another group of cases with the somatic symptoms of disseminated sclerosis is characterized by a preponderance of the mental symptoms,

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these appearing early in the course of the disease, of unusual severity, and terminating in complete dementia.

This group is represented pathologically by two subdivisions :

1. An extreme development of the sclerotic process in the gray and white matter of the brain (cerebral type of disseminated sclerosis).
2. A mixed or combined form. Dementia paralytica and multiple sclerosis occurring in combination.

Both are very rare. The following case represents the combination form of the two diseases :

SUMMARY OF CASE.* A woman, aged fifty-three years, presenting the symptoms of multiple sclerosis followed by dementia : spastico-ataxic gait; nystagmus; ataxic and intention tremor of arms; impulsive laughter; syllabic speech; double optic neuritis; progressive mental enfeeblement, terminating in dementia; duration, eight years.

Autopsy. Thickening of the calvarium; opaque and thickened meninges; marked atrophy of the frontal lobes; granular ependymitis; dilatation of the ventricles; disseminated plaques of sclerosis in the brain, cerebellum, pons, medulla, and cord.

Histological characteristics of general paralysis and disseminated sclerosis.

Clinical Notes. R. M., aged fifty-three years, was admitted to the Montefiore Home for Chronic Invalids October 2, 1896. She was the mother of seven children, two dying in infancy of negative causes. No miscarriages; no history of a syphilitic affection was obtainable. Her symptoms date back four years, and had followed an exposure to cold, beginning with uncertainty and weakness in the legs, which had grown progressively weaker.

Her memory is very defective. This with considerable mental enfeeblement render an accurate previous history impossible.

On admission patient was awkward in all her movements; gait spastico-ataxic; pronounced static ataxia both with open and closed eyes; ataxia and weakness of both upper extremities; right hemiparesis; speech stammering and syllabic; impulsive laughter; facial expression one of stupid hilarity; sight subjectively not impaired; examination of the internal organs negative.

Ophthalmological Examination. (Dr. Oppenheimer.) Double optic neuritis passing into atrophy.

Note, June 14, 1897. (Examination by Dr. Joseph Fraenkel.)

Patient is good-natured, complacent, and inclined to hilarity; complains of occipital tenderness and pains in the upper and lower extremities. Subjectively vision is not impaired. General paræsthesia (numbness and pricking), especially in the extremities. Pupils are small; all reactions present. The lateral excursions of the eyeballs are somewhat limited. Distinct lateral nystagmus.

The lower extremities are paretic, and passive movements encounter resistance. Ataxia and awkwardness of both upper extremities, especially the right.

No objective sensory disturbances.

* Presentation of Clinical History and Specimens at a meeting of the New York Neurological Society, February 4, 1902.

The jaw-jerk and the tendon-jerks of the upper extremities are wanting. The knee-jerks are lively and equal on the two sides. Ankle-jerks feeble on both sides and easily exhaustible.

Epigastric and abdominal reflexes absent; no involvement of the sphincters.

Note, June 25, 1899 (Dr. Fraenkel). Patient good-humored, silly, hilarious. Memory very poor. Says she has never been ill in her life. Extreme mental deterioration. Speech is stammering, with now and then a trace of syllabification. She can neither walk nor stand, and a change of position in bed is effected with great difficulty. Lower limbs are held in a state of flexion. Occasional involuntary atactiform movements of the arms and head.

The eyeballs are constantly dancing, and all movements except downward are accompanied by nystagmus. Pupils of equal size, well shaped, and the reactions to light and accommodation are present. Examination of the vision is unsatisfactory, but no serious defect is present. Sense of smell and hearing are apparently preserved. Motor cranial nerves are normal.

The arms are weak, especially the right. No atrophy. Marked iaco-ordination on both sides. On lifting a glass of water to the lips a typical intention tremor is elicited. Tendon reflexes not elicitable. No objective signs obtained.

The lower extremities are parietic and held in semiflexion. Of good volume. No resistance to passive movements; on the contrary, a slight degree of hypotonia exists. Slight hyperæsthesia; otherwise sensation normal. The right knee-jerk is lively; the left absent. The right ankle-jerk is present; the left absent. Plantar reflex is present on the right, absent on the left.

The muscles of the trunk are normal. Epigastric reflex absent. Electrically the muscles of the legs present sluggish faradic response. Reaction to the galvanic current normal. Died December 28, 1900.

Post-mortem Examination. The autopsy was performed December 29, 1900, by Dr. Harlow Brooks. A small tuberculous focus in the left apex. Purulent bronchitis. Interstitial nephritis. On removing the calvarium the anterior portion corresponding to the frontal lobes was found considerably thickened and eburnated, measuring from one-half to three-quarters of an inch. The inner surface of this thickened area is uneven, presenting nodular outgrowths and irregularities. The dura mater is normal. Moderate hydrocephalus *externus ex vacuo*. The leptomeninges are opaque and thickened, especially over the anterior aspect of the brain.

The convolutions of the frontal lobes are narrow and atrophic, the fissures wide and flaring. The whole anterior half of the brain, including the frontal and parietal lobes, appears small and compressed in contrast to the posterior portion. The ventricular cavity is enlarged. Moderate granular ependymitis. The vessels of the circle of Willis are free from arteriosclerotic changes.

After treatment with Müller's fluid the brain and cord were divided into serial segments for the demonstration and localization of plaques of sclerosis. Yellowish plaques of sclerosis sharply demarcated from the normal brain substance were found in the centrum semiovale on both sides in the cerebral cortex and in the cerebellum. These with the exception of a more diffuse sclerosis of the walls of the posterior

horns of the lateral ventricles were small and insignificant. Plaques were also present in the pons and the medulla bordering on the fourth ventricle and dipping down along the raphe. The plaques in the cord were very numerous, chiefly in the cervical and dorsal regions, including both the gray and white matter, with a predilection for the posterior and lateral columns. A few scattered foci were also present in the lumbar cord.

Microscopic Examination. *Brain:* The cortex is the seat of extensive changes; the cellular elements are tightly packed together and the glia cells are increased. The normal markings of the cortex are effaced. Many of the ganglion cells are sclerosed and atrophied. In many areas

FIG. 1.

Frontal lobe. Showing loss of the cortical fibre-system. Weigert's method.

the leptomeninges are thickened and infiltrated with round cells. The cortical arterioles are thickened, with increase of their nuclei, and the capillaries are increased in number.

The perivascular and pericellular spaces are enlarged, many enormously so, containing pigment, fat-granules, and detritus.

By the Weigert method a considerable loss of the radiating and association fibres of the cortex was demonstrable in the frontal, temporal, and occipital lobes. (Fig. 1.) In these areas the tangential fibres were absent.

In the Rolandic area, however, the cell changes were less intense, the medullary network better preserved, and the tangential fibres were

demonstrable. (Fig. 2.) No excess of pigmentation of the cells was noted, and the corpora amylacea were few in number. The cortical vessels were thickened and increased in number, but no widespread vascular or perivascular inflammatory process was noted.

Cord: The plaques of sclerosis in the brain and cord were composed of a dense network of glia cells and fibrillæ, either matted together in a dense network or arranged in rows and whorls. In these areas when dense the medullated nerve fibres had disappeared. By special stains (Mallory and Van Gieson) many naked axis cylinders were found present.

On the periphery of many of the plaques tongues and prolongations composed of glia cells radiated into the normal cord substance. The vessels in the sclerosed areas were considerably thickened, and the ad-

FIG. 2.

Rolandic area. Cortical network well preserved. Weigert's method.

jacent pia mater was thickened and occasionally infiltrated with round cells. The ganglion cells were of normal configuration and structure. The anterior and posterior roots were normal. Except the moderate round-cell infiltration of the meninges bordering the foci of sclerosis, there were no evidences of an exudative or myelitic process.

By the Weigert-Pal method the sclerosed areas were white and sharply defined, as if cut out with a knife. (Figs. 3 and 4.)

By the Marchi method the sclerotic foci were found free from myelin globules or granule corpuscles.

In portions of the cord not the seat of sclerosis many fibres showed the characteristic reaction, staining intensely black. In other fibres

this degeneration was only partial, a semilunar or concentric ring enclosing a normal axis cylinder. These fibre degenerations were diffuse and nowhere limited to the tract systems.

In the midlumbar region symmetrical patches of sclerosis occupy the tips of both posterior horns, on one side encroaching on the tract of Lissauer and the posterior root zone (anatomical explanation of the absent knee-jerk and Achilles jerk on the left side). The cauda equina was normal. There were no systemic degenerations in the long tracts of the cord.

Optic Nerves and Chiasm: Transverse section of the optic nerves just below the chiasm revealed numerous islets of sclerosis, most numerous in the central portions of the nerve. The nerve sheath is moder-

FIG. 2

Kolaniak area. Sclerotic plaque. Weigert-Pal method.

ately thickened. The islets of sclerosis consist of a much thickened bloodvessel wall, considerably diminishing the calibre of the lumen and a dense perivascular sclerosis. Surrounding and radiating from this as a centre is a fine glia network. In and around these areas the nerve elements are absent or diminished in number (Weigert method).

No round-cell infiltration or other evidences of recent inflammation were discoverable. Numerous amyloid bodies. The centre of the nerve fibre appeared pale in comparison to the deeply stained periphery by the Pal method, but the evidences of nerve atrophy were moderate. The same changes—vascular sclerosis and perivascular gliosis—were noted in the nasal portion of the optic tracts as they merge into the

commissure. No sharply defined plaques of sclerosis homologous to those found in the brain and cord were noted.

The following is a list of the recorded cases of the combined form of the two diseases, with abstracts of the clinical histories and pathological findings. There are seven cases in all, six with autopsy:

CASE I. (Schultze').—Man, aged thirty-six years. Syphilitic infection. December, 1876, onset with frontal headaches, pains in the extremities, and intention tremor. Diplopia; paræsthesia in the arms

FIG. 4.

Sclerosis bordering lateral ventricle. Weigert-Pal method.

and legs. Severe vertiginous seizure in June, 1877. Later, epileptiform convulsions. Paresis of right arm. Pupils unequal; good reactions. Weakness of vision without objective findings. Transitory aphasic attacks. Appearance of mental symptoms in October, 1878: Delirious, screams, strikes those about him; later, delusions of grandeur, followed by apathy, progressively developing stupor. Hæmatoma auris. Died December, 1878.

Pathological Findings. Marked atrophy and narrowing of the convolutions of the brain. Dilatation of the ventricular cavity. Epen-

dyma granulations. Systemic degeneration of the pyramidal tracts and columns of Goll. Multiple disseminated plaques of sclerosis in cord. Uniform thickening of the walls of bloodvessels and increase of the connective-tissue elements throughout the brain and cord.

CASE II. (Claus²).—Man, aged twenty-three years. Grandfather insane; father n tabetic. Early symptom, uncertain gait. Diminution of sight and hearing, followed by evidences of mental change. Articulation disturbed, but speech not scanning. The ataxia increased. Remittent palsy of the legs. Progressive mental deterioration. Diagnosis: general paralysis of the insane.

Autopsy. Thickening of pia arachnoid, especially over the frontal and parietal lobes. Stripping the pia over this area attended by loss of substance. The frontal convolutions are narrow and atrophic, fissures flaring. Dilatation of the ventricular cavity. Disseminated patches of sclerosis in the brain, brain stem, and cord.

CASE III. (Siemens³).—Woman, aged twenty-two years; single; hereditary taint; venereal infection. Admitted to the asylum in the summer of 1876 with acute mania. During this attack, which lasted six months, it was noticed that the legs were handled clumsily. Later, mental enfeeblement, scanning speech, intention tremor, and ataxia. No nystagmus. Epileptiform attacks of short duration. In July, 1878, a relapse of the mania, lasting four months, followed by dementia. Pupils not mentioned.

Autopsy. Leptomeningitis and atrophy of the frontal convolutions. Partial sclerosis of the cerebellum. Disseminated plaques of sclerosis in the cord, none in the brain.

CASE IV. (Zacher⁴).—Man, aged fifty-three years. In the autumn of 1878 a fall, followed by headaches and backaches. A few months later excitable and irritable. Failure of memory. Admitted to the asylum July, 1879. Pupils unequal; reactions present. Articulation difficult. Ataxic gait. Sensation normal. Knee-jerks +. Marked dementia. Delusions of grandeur. Intention tremor. Epileptiform attacks. Speech inarticulate. Marked tremor of body.

Autopsy. Atrophy of convolutions, especially over frontal lobes. Ventricle dilated. Vessels of cortex are increased in number. Walls thickened. Multiple plaques of sclerosis in the cord.

CASE V. (Otto⁵).—Man, aged thirty-five years. Onset with progressive amblyopia, weakness and uncertainty of legs. Later, ataxia of arms and legs. Optic atrophy. Nystagmus. Scanning speech. Increase of the tremor and ataxia. Exaggerated tendon reflexes. Disturbance of the tactile sense. Duration of the above symptoms eight years, followed by progressive mental failure terminating in dementia. Duration of the disease, twelve and a half years. Pupils equal and reactions present throughout.

Autopsy. Pachymeningitis interna hemorrhagica over the frontal region. Pia arachnoid thickened. Marked atrophy of the convolutions of the frontal and parietal lobes. Numerous foci of disseminated sclerosis in the brain and cord. Sclerosis of walls of ventricle of cartilaginous consistency.

CASE VI. (Otto⁶).—Man, aged twenty-seven years. Early symptoms: Vertigo, headache, and dimness of vision. Later, ataxia, nystagmus, intention tremor, scanning speech. Failure of memory. Dementia.

Autopsy. Thickening of the skull over the frontal region. Atrophy of the convolutions. Disseminated plaques of sclerosis in the brain, stem, and cord.

CASE VII. (Petroff *).—Without autopsy.

Clinical Study. Dementia paralytica and multiple sclerosis. Man, aged forty-seven years. No alcoholic excesses; no syphilis. Onset October, 1896, following a fall. Difficulty in articulation. Occasional attacks of mental confusion. August, 1899, ataxia and intention tremor of the arms. Walking almost impossible. Tendon reflexes exaggerated. Speech scanning. Mentally confused and dull. Incontinence of urine and feces. Can neither stand nor walk. No nystagmus. Optic disks normal. In January, 1900, attacks of depression and excitement. Tremulous speech. Tremor of the tongue and face. Ankle-clonus. Pupils normal. In August, 1900, delusions of grandeur. He is the Kaiser, has much money, is wealthy, etc. Dementia is pronounced. Speech disturbance is a combined scanning with a *silben stolpern*.

REMARKS. The present case is put on record as representing the combination form of the two diseases, multiple sclerosis and dementia paralytica.

Although the symptoms of disseminated sclerosis were not apparent until the forty-fifth year, the cerebrospinal distribution of the lesions and their macroscopic and microscopic features were identical with those of the juvenile form, while the extent, character, and severity of the cortical degenerative process would correspond only to that observed in general paralysis of the insane.

Premature senility might be suggested as a possible basis for the mental symptoms and cortical atrophy.

The age of the patient (fifty years) almost precludes this theory. Premature senility is, however, rarely observed; but even this unusual occurrence could be excluded by the absence of the usual senile changes in the brain—i. e., arteriosclerosis of the circle of Willis, encephalomalacia, pigmentation of the ganglion cells, corpora amylacea.

Diagnosis. The diagnostic difficulties encountered in such cases are considerable.

In the earlier stages of multiple sclerosis and dementia paralytica a certain resemblance is not infrequent, as many of the symptoms are common to both. Thus apoplecticiform and epileptiform attacks occur frequently in both affections.

Systemic degenerations in the posterior and lateral columns, so frequent in paresis, produce symptoms similar to those following a development of plaques in the same areas.

The nystagmus, scanning speech, and intention tremor when typically present leave no room for doubt.

But these important symptoms of multiple sclerosis are sometimes absent, or occurring in an atypical form may simulate the hand tremor

and speech disturbance of paresis. On the other hand, typical nystagmus and intention tremor are rarely observed in paresis.

The Argyll-Robertson pupil if present may justly be interpreted to indicate paresis—with some reservation, however, as this symptom is rarely met with in multiple sclerosis. Uhthoff⁷ in an analysis of one hundred cases of *sclérose en plaques* found the Argyll-Robertson pupil once, and in four cases myosis with sluggish light reactions. In well-marked cases of either affection errors in diagnosis would hardly be possible.

When the two diseases are combined the diagnostic difficulties offered are very great. In none of the recorded cases with autopsy was the combination form recognized clinically.

Usually in the earlier stages the condition was diagnosed as multiple sclerosis, but with the advancement of the mental symptoms the diagnosis of paresis was substituted.

It has happened that the clinical picture from the first has been that of paresis, the plaques of sclerosis being unsuspected until demonstrated by autopsy.

Cerebral Type of Multiple Sclerosis. In the advanced cases of pure disseminated sclerosis the lighter grades of dementia are not uncommon; this is usually attributed to an increase of the sclerotic process in the brain.

Some cases show early in their course symptoms of mental failure which advances rapidly to complete dementia. This is caused by a rapid extension of the sclerotic process in the brain (cerebral type of multiple sclerosis).

In such cases hallucinations, delusions, maniacal outbursts, attacks of melancholia, and delusions of grandeur are not uncommon. Here the pathological lesion consists of an extensive dissemination of the plaques in both the gray and white matter of the brain coupled with more diffuse sclerotic changes. Cases of this description are not common, and have been recorded by Schüle,⁸ Jolly,⁹ Greiff.¹⁰ Cases somewhat similar, but differing in that the sclerosis was diffuse, involving the brain *in toto*, have been recorded by Kelp¹¹ and Schüle.¹²

Cortical Gliosis; Optic Atrophy; Posterior Column Degeneration. Another group of cases described by Fürstner and Stuhlinger¹³ is of interest as bearing on the types under consideration. Their description is based on four cases having a similar pathology and symptomatology.

The essential pathological change consists in a diffuse gliosis of the marginal layer of the cortex. The glia proliferation is not confined strictly to this layer, but projects above the surface as small, rounded prominences (grauula and tubera), and dips down into the second and even third layer of cells.

The tubera often contain small cysts, and minute depressions are

seen in the cortex as a result of this cystic degeneration. The gliosis is more pronounced over the frontoparietal regions of the brain.

The cells of the cerebral cortex are fairly well preserved; where the process dips down among them they are compressed and undergo atrophy. The overlying pia is sclerosed and the skull is thickened.

In addition to the cortical changes, in all the cases there was optic atrophy and systemic degeneration in the posterior columns of the cord. The authors refer the gliosis to lymphocytes originating from the bloodvessels.

In all the cases no hereditary taint was traceable, with evidences of a defective nervous system in early life (imbecility, intellectual weakness, and in one case epilepsy).

In two of the cases the symptoms of amaurosis and paresis preceded cerebral symptoms by a considerable time.

The speech disturbance resembled that of paresis with a scanning element. Facial, tongue, and hand tremors were present.

Mentally the picture was that of a progressive dementia, lacking the intensity, however, of the paralytic dementia.

Although this condition clinically would simulate most closely a dementia paralytica complicated by paresis and optic atrophy, it might well resemble the groups of cases under discussion, and deserves to be recalled and emphasized in this relation.

To summarize briefly, a clinical picture characterized by a progressive mental enfeeblement, manifesting during its course other symptoms of mental alienation, as mania, melancholia, hallucinations, and delusions, and accompanied by the somatic signs of multiple sclerosis, may be caused by the following pathological changes:

An extreme cerebral manifestation of the sclerotic process, of the same nature histologically as the disseminated plaques in the cord.

A combination of the two diseases, general paralysis of the insane and multiple sclerosis, in the same subject.

The diffuse gliosis of the cortex with optic atrophy and degeneration of the posterior columns also arises for consideration in the differential diagnosis.

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TUMORS OF THE SIGMOID FLEXURE.¹

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FOR a long time my attention has been directed to tumors situated in the *left iliac fossa*—that is to say, of the *sigmoid flexure*—because occasionally there are to be found in this region palpable tumors the character of which is not easily recognized. May it therefore be permissible to give you a general review of tumors of this region? The most frequent occurrence of such tumors, which are *fecal accumulations*, present but small difficulty in diagnosis. They are easily recognized because of their comparative softness and because they not only occupy the descending colon, but also the rectum, where they may be easily felt by the examining finger. If in all doubtful cases you make repeated examinations after having thoroughly cleansed the bowels by laxatives or enemata, you will probably not be subjected to mistakes. The diagnosis becomes much more difficult with the *continuous presence of a tumor*. We must take under consideration intra-intestinal and extra-intestinal tumors, which may arise either from the pelvis, peritoneum, retroperitoneum, or the genito-urinary organs. The tumors may be new-growths, hard, or may be brought about by inflammatory swellings or by an abscess.

Of the *solid tumors* the most frequent are the carcinomata of the sigmoid. I say of the sigmoid, because I will only speak of the carcinomata confined to the sigmoid flexure and not of those of the rectum, which may extend upward. The recognition of the latter is not difficult when the physician, as a duty, will examine the rectum in every case where the symptoms point to disease of this part. It is not necessary for me to emphasize the latter point before you, members of this society, but rather to the general practitioner who frequently fails to do this. These cases are common enough where patients for a long time complain of dyspeptic symptoms and so-called hemorrhoidal bleeding, without the attending physician deeming it worth while to make

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