

Psychiatrisch-Neurologische Wochenschrift

(No. 8, May 20, 1905.)

1. The Etiology of Dementia Præcox. GREGOR STEINER.

1. *Etiology of Dementia Præcox*.—The author, after outlining Kraepelin's views on the subject, suggests the possibility that luetic infection may be an important etiological factor. In twenty-five cases of dementia præcox four gave a certain history of specific infection. If subsequent investigation should prove this relationship it would associate general paresis with dementia præcox, with which it has already many points in common.

(No. 9, May 27, 1905.)

1. The Question of a Medical Commission in the Care of Insane, Epileptics and Idiots. H. E. SCHWABE.

Medical Commission.—A paper dealing with the legal questions involved in the duties of a commission, and having no clinical interest.

(No. 10, June 3, 1905.)

1. The "Not Insane" in the Prussian Asylum Statistics. GRUNAU.

1. *"Not Insane" in Prussia*.—An article having only local interest, being a discussion of the bearing of the number not insane upon the percentage of recoveries in Prussian institutions, where it seems they were included in the total population and then added in with those discharged recovered.

(No. 11, June 10, 1905.)

1. The Parisian Opinion of Princess Louise of Coburg.

1. *Princess Louise*.—The opinion of Drs. Dubrisson and Magnan as to whether she should be confined in an institution and as to her sanity. They concluded her to be sane and not to require confinement.

(No. 12, June 17, 1905.)

1. The Evolution of German Psychiatry in the Beginning of the Nineteenth Century. MÖUKEMÖLLER.

1. *German Psychiatry*.—An article of only historic interest. (Continued.)

(No. 13, June 24, 1905.)

- 1.
- German Psychiatry*
- .—(Continued.)

(No. 14, July 1, 1905.)

- 1.
- German Psychiatry*
- .—(Continued.)

(No. 15, July 8, 1905.)

1. Opinion on the Capacity to testify of the Weak-minded. FRITZ HOPPE.

2. Contribution to "No Restraint." KERRIS.

3. The Evolution of German Psychiatry in the Beginning of the Nineteenth Century. MÖUKEMÖLLER.

- 1.
- Testimony of Weak-minded*
- .—A paper of purely legal bearing.

2. *"No Restraint"*.—A short paper in which the author discusses the influences of the environment of a modern hospital for the insane, the nurses, a more general understanding of insanity, and the like, that has created an atmosphere which makes restraint unnecessary.

- 3.
- German Psychiatry*
- .—(Concluded.)

WHITE.

Brain

(Vol. 27, 1904, No. 108, Winter, 1904.)

1. On the Spinal Cord Degenerations in Anemia. J. MICHELL CLARKE.

2. System Lesions of the Posterior Columns in General Paralysis, and their Bearing on the Point of Origin of Tabes Dorsalis. DAVID ORR and R. G. ROWS.

3. False Localizing Signs of Intracranial Tumor. JAMES COLLIER.

4. A Study of the Emotions. W. H. B. STODDART.

5. Symptomatology of Cerebellar Tumors. A Study of Forty Cases. J. GRAINGER STEWART.

1. *Spinal Cord Degenerations in Anemia.*—The author speaks of the unsatisfactory state of the present-day knowledge of the relationship of anemia to the degenerations of the spinal cord as comprised under well-known clinical groups. He reports the history of four patients with autopsy findings and the microscopical lesions observed. His series of cases showed that the spinal degenerations which occur as a secondary change in profound anemia begin in the posterior columns, first in the cervical and upper dorsal regions of the cord. The most striking clinical features are paresthesiæ of various kinds, such as numbness, tingling, feelings of pricking, or of pins and needles, first appearing in the arms and legs. Later the hand and forearms may be affected, when a common complaint is that numbness or deadness of the fingers renders fine movements difficult. These paresthesiæ vary much in intensity during the course of the illness. Objective disturbance of sensation is not marked, but in the late stages may consist of some deficient appreciation of all forms of sensation over the lower extremities. After these sensory symptoms weakness in the legs may follow, often in the sense that they feel heavy, and later still both arms and legs may be weak. A frequent and often troublesome symptom is twitching of the muscles, especially of the legs, at night. The deep reflexes are present, ankle clonus is not obtained, and the plantar reflex, flexor in type, is sometimes difficult to get. The knee-jerk was absent toward the end of life in Case II, but he could only be examined as he lay in bed. There is no ataxy and no incontinence of urine. The cases which he described form a fairly distinct group, and the changes in the cord occur in such a way as to show that they result from anemia, and from forms of anemia due to blood destruction. Though pernicious anemia is one form that causes these cord lesions, it is by no means the only one. He is here taking pernicious anemia in the ordinary sense of the term, though there is so much difference of opinion as to what actually constitutes pernicious anemia that it is difficult to avoid confusion. In favor of the point that it is only when anemia is due to blood destruction that these cord changes follow is the striking fact that they are not seen in chlorosis, of however long standing. A possible view of the pathology of these cases seems to the author to be that of parenchymatous degeneration due to some toxin, possibly set free in the course of a pathological blood destruction and, therefore, hemolytic in origin. More extended observations on the blood and on the precise character of the anemia would tend to elucidate the nature of the process. In this particular group the cord changes often follow on so long a period of anemia that it is difficult to suppose that the lesion is other than a secondary one, although it is possible that the cord changes might be due to the continued action of the same cause that has already produced anemia, just as in those cases of cord degeneration and anemia described by Dr. R. Russell and Dr. J. Taylor they suppose that both conditions are due to the same toxin.

Without laying undue stress on the anatomical characters of these cases, it seems to the author that there are good grounds, both clinical and pathological, for separating them from similar forms of degeneration of the cord, and that these are: (1) The presence of decided or severe anemia as the first, or sometimes the only, sign of illness; preceding any evidence of spinal cord lesion. (2) Clinically, symptoms and signs of disease of the cord are never very pronounced, and they may be absent, though possibly in most cases are present when looked for. (3) Anatomically, the lesion is generally confined to the posterior columns, and if it affects the lateral columns only, does so to a limited extent. (4) It differs in extent and distribution from sub-acute combined sclerosis or diffuse degeneration of the cord in the following ways, according as the disease

in the latter case is slight or severe. (a) In the milder cases of diffuse degeneration the degenerations are more precisely limited to the neuron systems of the cords; thus, in two cases under his own observation the columns of Goll were diseased from the lumbar region upward to the medulla, and the crossed pyramidal and dorsal cerebellar tracts were similarly affected, whereas in (b) the more severe cases the degeneration is both more extensive than in the cases considered in this paper, and also affects the periphery of the cord most, and appears to spread thence inward at any rate in the lateral and anterior columns. On the other hand, it must be admitted that the microscopical details of the degeneration process appear to be similar in both groups, and further that there are cases which occupy an intermediate position, in which the cord changes are associated with profound anemia, and are also extensive in distribution. It seems to the author that the chief difficulty lies in the nature of these intermediate cases.

In conclusion, although the evidence is not sufficient for a final classification, the author thinks that a step in advance might be made by separating into two distinct groups: (1) The cases of pronounced anemia with lesions in the spinal cord of the character described in this paper, and with slight or absent clinical features of the disease; and (2) those of subacute combined degeneration of the cord in which there is no anemia, or if it is present it is late, and consecutive to the appearance of symptoms of cord disease, and in which also the pathological changes in the cord are more extensive and their symptoms pronounced, following more or less closely the course outlined by Drs. Russell, Batten and Collier. In support of this division it may be mentioned that in fifty cases of the latter kind collected by Drs. Putnam and Taylor, in only seven was there decided anemia. Those cases which seem to combine the features of the two groups, in presenting both profound anemia and widespread cord degeneration, the author would propose to leave provisionally in a class by themselves pending further investigation.

2. *Posterior Column Degeneration in General Paresis.*—The authors contribute a lengthy study on fiber changes found in the columns of Goll and Burdach in paresis, and make some interesting comments on the relation of that disease to tabes on the score of these posterior degenerations. In the majority of cases of paresis there was no posterior column sclerosis, but in those in which degeneration was found it was characteristic that it always began in one place, *i. e.*, at the entrance of the posterior roots into the cord, and from that spot spread to the fiber terminations. The authors say with reference to the pathological picture that there is (1) degeneration of the internal division of the sensory roots in their intramedullary path, commencing at the point of entrance into the cord and following the usual anatomical course of the fibers of the cord. (2) The collaterals and terminals of the sensory system, which pass into the gray matter, share in the degenerative process. (3) Complete integrity of the outer division of the sensory root, Lissauer's tract. (4) The long fibers which were affected in the root-entry zone in the lumbar region could be followed into Goll's column in the cervical region.

Obersteiner's ring was the site of election of the attack. Here, as is well known, the neurilemma sheath is lost, and the hypothesis is suggested that the bare nerve fiber is subjected to the action of a toxin-laden lymph stream which flows toward the cord in the perineural sheath. The poisoning results in a primary degeneration of the nerve fiber and a consecutive atrophy of the myeline sheath—the axis cylinder may ultimately give way secondarily, the fiber degenerating from the point of injury centrifugally to its termination. The authors contrast their view with that of Nageotte, who assumes Wallerian degenerative process to be fundamental in posterior column sclerosis. Tabes is a system disease, they conclude, which begins as a parenchymatous degeneration of the sensory protoneurones, starting at the point where the neurilemma is lost.

3. *False Localizing Signs of Intracranial Tumor.*—Dr. Collier has based his paper on the study of 161 cases of intracranial tumor that have come to autopsy in the National Hospital in the ten years between 1894 and 1904. The author seeks to show: (1) That local signs appearing late in the course of cranial tumor, where general signs have pre-existed, are often of false portent. (2) The relative frequency with which local signs have been due in this series of cases to the presence of vascular lesions, meningitis, hydrocephalus, local spreading edema of the brain, secondary deposits of new growth, and posterior degeneration. (3) That the absence of usually accepted local signs during the early days of illness in intracranial tumor is in itself a most important localizing indication, confining the disease to the supratentorial region. (4) That true localizing signs at one time present may later become concealed or undemonstrable, owing to the development of other signs, and that in cases which come under observation for the first time late in the disease diagnosis may be difficult, erroneous, or impossible.

The relative frequency with which the different parts of the brain were the seat of new growth in this series of cases was as follows: Frontal 24, central 20, parietal 7, occipital 3, temporo-sphenoidal 12, centrum ovale 17, basal ganglia 14, brain stem and pons 22, cerebellum 26, other situations 16.

As illustrative of what he would convey as late localizing signs, he appends a summary of a history wherein a late involvement of the left sixth, seventh and eighth cranial nerves, after fifteen months of general symptoms, was due to the pressure backward of a glioma in the left pre-frontal region. Localizing Jacksonian convulsions in another case were due to general ventricular distention. False localizing signs were found in 12.5 per cent. of all his cases. In two instances the false signs were due to hemorrhage and thrombosis respectively; in the rest the false signs were to be attributed to the indirect intracranial results of intracranial new growths. They were met with in 13 per cent. of the supratentorial tumors, and only twice in fifty-four cases of subtentorial tumors. He reports the following localizing signs, occurring either singly or in combination, as false signs: Paralysis of cranial nerves; olfactory (once), hemianopsia (two cases), third (two cases), fifth (two cases), sixth (twelve cases), seventh (two cases), eighth (two cases); ninth, tenth and twelfth were not indirectly affected.

He submits, therefore, that the occurrence of local convulsion of hemi-epilepsy and of general convulsion, when presenting for the first time, long after the general signs of intracranial growth have appeared, is to be disregarded as a localizing sign. It is the result of secondary hydrocephalus, and the latter may result no matter where the growth may be situated within the skull. Slight bilateral spasticity is not infrequently an indication of the existence of such ventricular distension. The author further considers the symptoms due to other lesions co-existing with a neoplasm, such as meningitis, vascular lesions, local spreading edema. The paper requires reading in full to be fully appreciated.

4. *A Study of the Emotions.*—Dr. Stoddart in this paper, in first speaking of the physical basis of the emotions, submits two propositions as capable of demonstration: (1) That an emotion is a sensation-complex, its component sensations being entirely derived from a complex, usually involuntary, motor response to a percept or idea of some situation or incident. (2) That the motor paths subserving the function of this involuntary response are those of the primitive nervous system, viz.: the cortico-rubral system of fibers and the rubro-spinal tract. He derives certain proof from the use of the plethysmograph, the sphymograph, pneumograph, dynamometer and automatograph, and categorically states his conclusions. With reference to the second point he summarizes the neural process which takes place when an emotion occurs as follows: Starting from the stage at which

a sensation is registered in one of the projection areas, or a percept or idea formed in one of the association areas of the cortex, an impulse is transmitted to the red nucleus by way of the cortico-rubral fibers, thence to the large motor cells of the lowest level by way of Monakow's rubro-spinal (and presumably rubro-bulbar) fibers of the pristine motor system, and thence to the muscles of expression. Contraction of these muscles upon their spindles effects the transmission of the muscle-sensations to the cortex by way of the ordinary sensory paths; and it is the particular combination of these sensations among themselves and their vaso-motor sensations which determine the given affective or emotional tone. In a second section he deals with the pathology of the emotions, discussing superficially the excess of emotional reaction and deficiency of emotional reaction, summarizing his conclusions as follows: Excess or defect of emotional reaction may be dependent upon excess or defect of sensation, or upon excess or defect of perception. Excess of emotional reaction may also depend upon an abnormal tendency of motor impulses to be transmitted via the pristine emotion-arousing tracts of the nervous system, and defect of emotional reaction may further be due to fixation of emotion-arousing musculature.

5. *Cerebellar Tumors. Symptomatology.*—Drs. Stewart and Holmes, in analyzing forty cases of cerebellar tumor, have contributed a small monograph on the subject. In twenty-two instances the autopsical report was utilized in the analysis. They have divided their study into two sections, dealing with (a) tumors within and limited to the substance of the cerebellum, and (b) those in which the growth was extracerebellar, or those tumors lying in the posterior cranial fossa in the angle between the pons varolii and the cerebellum, compressing but not invading either. In speaking of the general symptoms attention is called to headache, vomiting and optic neuritis. The *headache* is usually present early and constantly in both types of tumor. It is similar in character to the headache of other intracranial growths, but its situation may be suggestive. In intracerebellar growths the pain is most intense occipitally and frequently circumscribes for long periods, or it may radiate down the back of the neck or between the shoulders. Frontal and retro-ocular headaches are next most frequent. In extracerebellar tumors the pain is almost always occipital and may radiate down the neck. The headaches are apt to be extremely severe. *Vomiting* is almost invariably present in some stage of cerebellar and extracerebellar tumors. *Optic neuritis* is a constant accompaniment of intracerebellar neoplasm. It comes on early and is often intense, and disproportionately so in character. Transient blinding attacks are common. *Vertigo* is a constant symptom in both types, particularly in the extracerebellar forms. It is apt to be transient, a "giddy feeling" being characteristic of one type. There is often as another type a sense of displacement of external objects from the site of the lesion. Thus, if tumors are on the left side there is an apparent movement of objects from left to right. In intracerebellar tumors the subjective rotation of self was always from the side of the lesion to the healthy side, whereas in extracerebellar tumors the reverse condition was present. The authors regard this symptom of much diagnostic import. *Deafness* was present in practically all extracerebellar tumors and on the side of the tumor. It was a negligible factor in the other type. Tinnitus is fairly common in extracerebellar tumors, and is in the corresponding ear. Cranial nerve symptoms are common. The majority of cases of both types develop sometimes varying degrees of rectus palsy on the side of the disease. Squint and diplopia are often transitory, but are liable to recur, and more often present in extracerebellar tumors. Bilateral external rectus paresis is especially met with in tumors of the middle lobe of the cerebellum. *Pupils* are not markedly influenced, the optic neuritis influence being paramount. The third and fourth nerves are almost invariably in-

tact. Nystagmus is a constant symptom. Slow, deliberate jerking movements to the side of the lesion in looking in that direction, with gradual recession of the eye to the middle plane, are characteristic. Lateral and vertical movements show the nystagmus most markedly. *Facial paralysis* is not infrequent, chiefly due to extracerebellar pressure.

MOTOR SYMPTOMS.—Paresis in the limbs of the same side as the tumor is not infrequent. There is usually no accompanying organic rigidity. The trunk muscles particularly are involved. The extracerebellar tumors rarely give rise to such definite homolateral paresis, but may cause symptoms more indicative of the hemiplegic type from cortico-pontine pressure. *Atony* of the muscles is common. They are frequently flaccid and limp. *Ataxia*, a classical symptom, is a true dysmetria, the agnostic and antagonistic being incorrectly opposed or misjudged. It is a central ataxia, rather than a peripheral one. Ataxia is not increased on loss of vision. It is an ataxia the direct antithesis of an intention tremor, the muscles becoming firmer as the object is gained.

In cases of chronic course, or where the lesion has become latent, the ataxy is, as a rule, less definite. This is especially so when the condition has become complicated by hydrocephalus; then it may be less typical, and approximate to the intention-tremor type characteristic of disseminated sclerosis. This seems to occur most frequently in tumors involving the vermis. In disease of one lateral lobe the incoördination is typically present only in the homolateral limbs, but not infrequently there is in addition some degree of ataxy in the opposite limbs, though always less in degree. When the tumor is in the vermis, or extends into both lateral lobes, the incoördination is bilateral, but greater in the limbs of the side on which the lateral lobe is more affected, or it may be more pronounced on one or the other side as the effect of the tumor varies. The incoördination affects the arm more than the leg. The reeling gait would seem to suggest that it is greater in the lower extremities, yet on examination it is found that this is due in great measure to the irregular action of the trunk muscles. The author has often noted when the patient stands erect that an unnatural degree of alternate contraction of the erectors spinæ of the two sides occurs as he attempts to maintain or attain his proper balance. Patients who are so unsteady on their feet that they can hardly walk present a comparatively slight degree of incoördination in carrying out movements as they lie in bed. He has not been able to make out any constant difference between the ataxy which results from cerebellar disease and that met with in extracerebellar tumors. Some slight degree of incoördination generally persists for many months after the disease has been removed by operation, and may be one of the last signs to disappear. He has carefully investigated in a number of his cases the sign described by Babinski as pathognomic of cerebellar disease, to which he has given the name of *diadococinesia*. It consists in the inability of the patient accurately to perform rapid alternate movements with the homolateral limb, though the individual movements are easily possible. It is tested by requesting the patient to pronate and supinate the forearm in rapid succession; this can be quite naturally executed by the contralateral limb, but the homolateral arm is moved slowly, awkwardly and irregularly. This phenomenon seems forcibly to confirm the author's analysis of cerebellar ataxy—that it is a dysmetria or defective control of muscular action, with inaccurate combination of the component muscular contractions. It seems to him that this inability rapidly to repeat movements is dependent upon the defective coöperation of the muscles and their antagonists, due in part, at least, to the diminution of the reflex muscle tone described, and to the consequent failure of the antagonists to control the primary movement, owing to their lack of tone.

Attitude.—In many cases of unilateral cerebellar and extracerebellar tumors the head is held in more or less characteristic attitude. When the

patient sits or stands it is slightly flexed to the side of the lesion and rotated so that the chin is directed toward the opposite shoulder, and the occiput approximated to the point of the homolateral shoulder. Though very characteristic, this attitude is neither constant nor pathognomic. Occasionally in cerebellar growths the head is held in the reverse position, and this "cerebellar attitude" of the head is met with in cases of pontine, and, more rarely, of fore-brain neoplasms. It cannot, therefore, of itself be used as a positive sign of cerebellar disease or as a definite aid in its localization.

Gait.—The gait of cerebellar disease has been accurately described as drunken, staggering or reeling, but further analysis of its characteristics is necessary before it can be used as an aid in localization of the lesion. There appear to be two distinct components which characterize the gait peculiar to unilateral cerebellar disease: (1) A tendency to stagger and stumble to the side of the lesion; (2) a tendency to deviate from the desired course toward the side of the lesion. The stumbling is more or less characteristic; the patient when walking suddenly totters as though to save himself from falling, as if he were drawn by an unseen influence toward the side of the disease, but as a rule, he quickly recovers his balance and direction. He may occasionally stumble to the opposite side, but he does so less frequently and to a less extent. This stumbling is rarely of such a kind as to suggest that it is due to weakness or giving way of the homolateral leg.

Sensory Symptoms.—Alteration of cutaneous sensibility never occurs in cases of cerebellar disease except when the tumor involves the pons, either directly or indirectly, by pressure, or causes secondary softening.

Mental Condition.—Cerebellar growths do not seem to have any effect on the psychical functions *per se*, though any acute increase of intracranial pressure may cause a drowsy or stuporous state, as a rule of brief duration.

Tendon Reflexes.—The variability of these reflexes is one of the most striking signs of cerebellar lesions. In some cases they are increased, while in others they are diminished or lost, but any one case may present both extremes within short periods.

The author further considers the differential diagnosis in a complete and detailed manner, which precludes abstraction, as does also a discussion of the relation of the symptomatology of cerebellar disease in man to the experiments in animals. A full clinical summary is appended.

JELLIFFE.

Journal de Psychologie Normale et Pathologie

(Vol. 2, 1905, Jan.-Feb.)

1. Repeated Attacks of Motor Aphasia in a Morphinomaniac. ROY and JUQUELIER.
2. A Comparative Study of the Psychology of Certain Motor Manifestations Commonly Referred to as "Tics." DROMARD.
3. A Note Upon the Conscious Appreciation of Words in the Use of Language. LALANDE.

1. *Repeated Attacks of Motor Aphasia in a Morphinomaniac.*—The case reported is that of a woman, sixty-one years of age, who was addicted to the use of morphine (about 1 gramme a day) for twenty-eight years. She presented at no time the slightest evidence of any sort of organic lesion, unless a slight degree of cardio-vascular excitability were to be considered as such. Gradual withdrawal of the drug was successfully accomplished with the aid of psychotherapy. Five different times she had had attacks of motor aphasia, the last attack having been observed and closely studied by the writers. Typical in every respect, this motor aphasia