The paper is illustrated by coloured drawings, which do not, however, altogether meet the object of the authors.

R. M. S.


It is well to have a thorough-going and complete investigation of the fibre-anatomy of the basal ganglia, since speculation as to striatal function has often outrun anatomical facts, on which alone physiological theories must rest.

Relying largely on the experimental results of the Vogts and of Wilson, the author has prosecuted further researches by experimental methods in cats, dogs, and rabbits, and by the examination of certain human brains, including that of a normal newborn infant and of an eighth-month foetus.

His conclusions are too long to be conveniently summarized here, but among them may be mentioned the following:

There is a definite tractus striomesencephalicus ad substantiam nigrum, which arises chiefly in the head of the caudate, and is characterized by the fineness of its fibres;

The main constituents of the laminae medullares of the globus pallidus are striopallidal fibres;

The fibres of Forel's bundle (H1) are chiefly striopetal, but among them are some that are striofugal;

Forel's (H2) bundle contains mainly pallidofugal fibres, but some are striopetal;

The corpus Luysii is doubtfully connected with oral segments of putamen-caudate and globus pallidus;

There is a definite pallidofugal connexion between globus pallidus and posterior longitudinal bundle;

The red nucleus has a definite connexion with the corpus striatum by chiefly pallidofugal fibres, though some appear to be from the putamen-caudate;

There is no proof of any connexion between cortex and putamen-caudate by fibres passing in either direction, but in the cat the author found a cortico-pallidal relationship, degeneration being present in the globus pallidus after lesions of the frontal poles.

S. A. K. W.

NEUROPATHOLOGY.


In normal patients the number of cells in each successive cubic centimetre of fluid drawn off at lumbar puncture decreases very rapidly from the first to the fifth cubic centimetre, and thereafter more slowly. In a series of fifteen
cases with normal cell counts it was found that there were often six or eight times as many cells in the first cubic centimetre as there were in the tenth cubic centimetre. This decrease applied equally to the lymphocytes and the large mononuclear cells. A similar rapid decrease of cells was noted in two out of three cases in which pleocytosis had been induced by the injection of normal saline or of ‘hémostyl’ at a previous lumbar puncture. It was thought, however, that there might be a fallacy in results obtained from meningeal irritation produced in this way, as the site of injection (that is, the site of lumbar puncture) would naturally be the site of greatest irritation. To obviate this objection observations were made after the injection of air by lumbar puncture for the purpose of ventriculography. This was found to produce an intense pleocytosis, rising as high as 30,000 per cubic millimetre, and consisting chiefly of polymorphonuclear cells. In these experiments the difference in the number of cells in the earlier and later drops of fluid was very much less, and in fact was in many cases negligible, but a fall in polymorphonuclear cells was noted in all, and in total cell count in eight out of the ten cases. The greatest fall, and in fact the only considerable one, was from 30,000 polymorphonuclear cells in the first cubic centimetre to 1,800 in the thirty-sixth cubic centimetre. In none of the other cases did the cells fall by as much as half their original figure. It is noteworthy that in the two cases in which the least pleocytosis was induced there was no drop in the total cell count between the first and the twentieth cubic centimetre.

The authors plead for a more rigorous technique in cell counting, especially that the cells should always be counted in the first 2 c.c. of fluid withdrawn. The value of this procedure does not, however, appear to be proved from their paper, for the enumeration of these cells which, on their theory, may have gravitated to the lumbar cul-de-sac from distant parts of the subarachnoid space, would surely not give a more accurate index of the degree of meningeal inflammation than is obtained by the more usual procedure of counting the cells distributed, more or less evenly, through 5 or 10 c.c. of fluid.

J. G. GREENFIELD.


This paper is a confirmation of the observation of Ayala (Zeits. f. d. g. Neurol. u. Psychiat., 1923, lxxxiv), that the pressure of cerebrospinal fluid drops in the course of lumbar puncture much more rapidly in cases of cerebral tumour than in those of serous meningitis and hydrocephalus. An index of the rate of fall was obtained by Ayala by multiplying the number of cubic centimetres of cerebrospinal fluid removed (Q) by the final pressure in centimetres of H₂O (F) and dividing by the initial pressure (I), thus: $\frac{Q \times F}{I}$. This index was found by Ayala to vary as a general rule between 2.55 and 4.55 in cerebral tumours and between 7 and 10 in serous meningitis. But in five out of eighteen cases of cerebral tumour he obtained rather higher indices suggesting serous
meningitis. Balduzzi, in a series of fifteen cases of cerebral tumour, found the index to vary from 1.4 to 5.5, whereas in eight cases of serous meningitis and two of tubercular meningitis (in some of which the patients were punctured on several occasions) the index always lay between 6 and 11.6. He considers, therefore, that values below 5.5 are indicative of cerebral tumour and above 6.5 of serous meningitis. Intermediate indices between 5.5 and 6.5 are of no diagnostic value. He considers the determination of cerebrospinal fluid pressure in cerebral tumour to be of service, not only for the diagnostic purpose of obtaining this index, but also as a safeguard against removing too much fluid, for if fluid escapes slowly and the pressure is not allowed to fall below the normal there is little risk of unpleasant sequelae.

J. G. Greenfield.


Among many writers who recommend that in lumbar puncture the patient's neck should be bent as far forwards as possible there are very few who warn the operator that readings of fluid pressure taken in this position are fallacious, as strong neck flexion not only embarrasses respiration but tends also to obstruct the jugular veins, and thus greatly raises the intracranial venous pressure on which the cerebrospinal fluid pressure depends. Van Loon has found that in some patients forced forward flexion of the neck may raise a normal fluid pressure of 100 or 140 mm. to 350 or 400 mm.; turning the head to one or other side is also found to raise the fluid pressure, though not to the same extent. He therefore recommends that the patient should be allowed to straighten the body and neck before the pressure is measured.

Very high pressures can be obtained by a combination of neck flexion and constriction of the neck veins by a rubber band or the arm band of a Riva Roeci sphygmomanometer. When these measures are adopted it is possible to "squeeze out" large quantities of cerebrospinal fluid, up to 120 c.c., without causing any unpleasant sequela such as headache or collapse. This the author attributes to the fact that the cerebrospinal fluid pressure was always kept at the same level, or slightly raised during the lumbar puncture. He considers that this method of obtaining large quantities of fluid offers several therapeutic advantages, as intrathecal remedies can be given in a much more dilute form, and are more likely to reach rapidly the intracranial parts of the subarachnoid space. A case of meningoeoeal meningitis treated in this way made an unusually rapid and complete recovery.

J. G. Greenfield.


In an analysis of the sugar estimations in 421 spinal fluids the authors contribute determinations in the normal person, in epidemic encephalitis, in general paralysis (treated and untreated), in dementia praecox, in manic-depressive insanity, and in various miscellaneous conditions. The Benedict-Österberg method for urine, as modified by the authors for the spinal fluid,
was used, together with a control in each case by a determination by the Folin-Wu sugar method.

The normal range was found to be from 50 to 65 mgm. per 100 c.c. of spinal fluid. In epidemic encephalitis the average figure was 82 mgm. per 100 c.c.—an increase which the authors consider of diagnostic value. In twenty-one cases of dementia precox the spinal fluid sugar showed an average of 80.1 mgm. per 100 c.c., but high readings occurred less often than in epidemic encephalitis. In both treated and untreated general paralysis the spinal fluid sugar lies within normal limits, and no noteworthy change occurs in manic-depressive insanity.

R. M. S.

[99] The manner of infection of disseminated sclerosis (Zur Entstehung der multiplen Sclerose).—B EH R. M ü n c h. med. W o c h., 1924, lxxi, 633.

That disseminated sclerosis is infective in origin can no longer be denied; the primary pathological change is not, as formerly thought, an overgrowth of glia, but alterations in and around the blood vessels, viz., hyperemia, infiltration of the vessel walls with lymphocytes and plasma cells, and capillary hemorrhages; with these changes are associated changes in the adjacent nerve elements, especially the myelin sheaths; circumscribed areas of lymphocytic infiltration have also been demonstrated in the meninges. Several investigators have succeeded by the method of Kuhn and Steiner in inoculating dogs and guineapigs with material from a recent focus in the human brain and producing nervous symptoms which have a great similarity to those of human disseminated sclerosis; further, Kuhn and Steiner have succeeded in transmitting the disease thus produced from animal to animal.

Assuming, then, that the disease is inflammatory, in what way does the causal organism gain access to the nervous system?

In a large proportion of cases the earliest symptoms are visual, and even in cases in which there have been no visual symptoms the optic discs are frequently found partly atrophied when other symptoms appear. Out of 144 cases at the Kiel clinic there were definite changes in the discs in 102; thirty-five of these cases began with visual symptoms and fourteen others certainly showed pallor of the discs before any symptoms came on, so that in at least forty-nine cases (33 per cent.) the first patch of which there was any evidence was in an optic nerve. It is reasonable to infer that the earliest lesion is frequently in an optic nerve. Now, patches in the optic tracts, which have the same structure, are rare; it is therefore unlikely that the frequent early involvement of the nerve depends on any special susceptibility or chemical affinity of its structure for the causal organism; it must depend in some way on its anatomical relationships.

Again, the offending microbe may reach the site of inflammation by one of three means—by the blood-stream, by the lymph channels, or by direct spread from an infected site in contact with the outer world. The fact that, although the inflammation in the optic nerve is an acute process, years may elapse between its occurrence and the appearance of any signs of diffusion of the infection, is strongly against the theory of invasion via the blood or
lymph. But no part of the central nervous system is so accessible by direct spread from without as the optic nerves; they are separated from the mucous membrane of the posterior ethmoidal cells only by (1) a very thin plate of bone pierced by numerous vessels and nerves and sometimes even absent, and (2) very vascular connective tissue containing many lymph spaces.

For these reasons Behr thought it possible that the infective organism might be present in the posterior ethmoidal cells, and he inoculated into the subdural space of dogs material from these cells in four cases of early disseminated sclerosis with retrobulbar neuritis. The first animal developed hyperaemia and swelling of its right optic disc a month after the inoculation, and within six months both discs were partly atrophic. The second showed hyperaemia of the right disc after four weeks, and subsequently pallor; eight months later hypotonia of all the limbs came on; this was thought to be of cerebellar origin. The third animal in seven months developed partial atrophy of both discs and left hemiplegia and a left oculomotor palsy. The fourth died suddenly six weeks after the inoculation. The brains of two of the dogs are now being examined microscopically, and a further communication is promised.

From the signs observed in these experimental animals it is apparent that multiple lesions occurred in them, and, as regards the ocular phenomena, Behr justly emphasizes their similarity to those which occur in the human subject. He argues that disseminated sclerosis is due to an organism which gains entrance to the central nervous system by the bony passages from the ethmoidal cells and that the organism may be present in this situation in healthy subjects and be harmless as long as it does not enter the cranium.

J. P. Martin.


The authors made a histological examination of the nervous system in two cases of myopathy and one of Thomsen's disease. (The age of the patients at death is not stated.) They found: (1) Degeneration of the cells of the putamen with the formation of brown pigment, differing altogether from the normal intracellular lipochrome of nerve cells. This pigment was also found round the vessels and in the neuroglial cells. In the globus pallidus there was degeneration of some of the large cells into round deeply staining masses. This cellular degeneration was associated with neuroglial overgrowth. These changes were much more marked in the case of Thomsen's disease than in the others. (2) The melanin-containing cells of the brain stem, especially those in the locus coeruleus, in the dorsal vago-accessory nucleus and in the formatio reticularis, also showed certain changes described as of three grades: (a) great swelling of the nucleus at the expense of the cell body; (b) atrophy of the cell; (c) disappearance of the cell and discharge of free pigment. In contrast with the cell groups mentioned, the substantia nigra showed no gross changes. (3) Cell changes in the infundibulo-hypothalamic region, in Reichert's substantia innominata, the melanin-free cells of the formatio reticularis, and the
dentate nucleus of the cerebellum. These changes consisted in cell atrophy with excess of lipochrome pigment and sometimes with vacuolization.

The authors consider these cell changes as 'abiotrophies' and as an integral part of the familial or hereditary disease. **J. G. Greenfield.**

[101] **The pathological anatomy of Huntington's chorea (Zur Frage der pathologischen Anatomic chronischer progressiver Chorea von Huntington).—SNESSAREW. Zeits. f. d. g. Neur. u. Psychiat., 1924, xei, 463.**

Among the pathological findings recorded by the author in a well-considered paper are the following: (1) heteropia of the ependyma and ependymal gliosis; (2) generalized hyperplasia of the glia, with evidence also of a reactive necrobiotic process (amoeboid glial cells, spongioplasmic elements, alterations of the gliosomes); (3) well-marked granular formations in glial cells and along glial fibrils; (4) granular collections in the ground-substance between the nerve elements, and numerous deposits of the corpora versicolorata of Siegert.

The important conclusion is drawn that to the hereditary factor of degeneration is added another factor of etiological moment, viz., one of a toxic character. Dr. Snessarew disagrees with any attempt to localize the morbid processes entirely or mainly in the corpus striatum, indicating that their complexity and widespread nature militate against any such supposition. He is content, on the other hand, to assign the hyperkinesis of progressive chorea to 'an affection of the corpus striatum,' without any effort to explain this statement.

**S. A. K. W.**


Professor Brouwer's case is that of a mentally deficient child, who died at the age of one year and nine months. Clinical details are lacking.

The cerebellum was very small, but the vermis was much better developed than the hemispheres. The pons, similarly, was small, whereas the medullary olives were more or less normal. In the cerebellum cortical changes were pronounced in lobus anterior and lobus simplex, viz., microgyria and poor myelinization. The nuclei tecti, globosi, and emboliformes were normal, while the dentates showed marked arrest of development. Only their dorsal part, which is 'old' and connected with the palaeocerebellum, was better formed. In the pons the middle cerebellar peduncles were atrophic; in fact, in Weigert-Pal preparations the whole sections were 'white,' with the exception of pyramidal and cerebro-cerebellar paths (frontopontine, temporopontine).

The case belongs to an unusual type of hypoplasia, the pontocerebellar tracts being implicated and the olivo-cerebellar practically intact. The author ascribes it to a process of congenital systematized degeneration.

**S. A. K. W.**

[103] **Hepatic function in postencephalitic Parkinsonism (Funzionalità epatica nel parkinsonismo post-encefalitico).—PAOLO OTTONELLO. La Diagnosi, 1924, iv, 1.**

This painstaking study is based on the investigation of 15 cases of chronic
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postencephalitic Parkinsonism, presenting a variety of clinical features otherwise (mainly unilateral in 2 cases, profound malnutrition in 1, psychical disorders in 3, polydipsia in 3, daily rise of temperature in 1, etc.). The following are the results of the examinations:

1. Liver increased in volume, 8 cases; diminished, 1; subicteric tint of skin, 1.
2. Urobilinuria, 6 cases; increase of reducing substances in urine, 5; glycosuria, 1.
3. Diminution of urea-nitrogen in every case, and of total nitrogen, 11 out of 13 examined.
4. Increase of ammonia-nitrogen and of amino-acid-nitrogen, 7 and 10 cases respectively.
5. Roch’s test. If 0·002 grm. methylene blue is given by mouth it should not appear in the urine if liver function is normal. It was present in 13 cases.
6. Roch and Schiff’s test. If 0·04 grm. sodium salicylate is similarly given, it should be dealt with by a normal liver and not appear in the urine. It was found in the urine in 10 cases.
7. Alimentary glycosuria, 5 cases.
8. Adrenal glycosuria (subcutaneous administration of 0·001 grm. adrenalin chloride), positive in 10 cases.
9. Galdi’s test of adipolytic function of liver. An increase of lipoids or of fatty acids or their derivatives in the blood serum will precipitate the globulin of the serum in the presence of glycerinated water. This indicates hepatic insufficiency, and is tested two hours after the administration of 50 grm. of butter. Result, positive in 6 cases out of 9.
10. Digestive haemoelasis (Widal) positive in 9 cases, doubtful in 4.
11. Experimental ammoniuria positive, 9 cases.

From a consideration of these findings the author concludes that inefficiency of liver function is a practically constant feature of postencephalitic Parkinsonism, but there is no parallelism between its degree and that of the neurological affection. He supposes that in the acute stages of the disease the liver is implicated just as it may be in any severe infection, that in due course its function becomes a dysfunction, which in its turn has a repercussion on the whole organism. Possibly, too, the diffuse nature of the inflammatory processes in the disease may lead to implication of postulated ‘vegetative’ centres in the brainstem (sialorrhoea, vasomotor disorders, tachypnoea, bradycardia, etc.), hence may also involve sympathetic centres regulating liver nutrition and activity.

S. A. K. W.


However marked at autopsy the liver cirrhosis in cases of Wilson’s disease or pseudosclerosis may be, the ordinary clinical picture of that disorder is never manifest during life. Special investigations are required to demonstrate
its presence. The author gives his results in the examination of a case belonging to the group above mentioned.

1. **Alimentary Glycosuria.**—After the administration of 30 grm. of galactose the urine withdrawn at the end of two hours showed reduction and rotation of the plane of polarization. The total amount of galactose found in the urine was 800 mgm. After the administration of 100 grm. of levulose the urine withdrawn at the end of one hour gave a distinct reaction to reduction tests; and these were slightly positive in the specimen taken after two hours. In all, 200 mgm. were obtained.

2. **Nitrogen Metabolism.**—In the urine of the patient with defective hepatic function an increase of ammonia and amino-acid nitrogen and a diminution of urea-nitrogen should be found. This was demonstrated by Dr. Ivens after the usual hospital diet. Further, if 10 to 20 grm. of amino-acid (glycocol, alanin, asparaginic acid) are given by the mouth, there should not, in normal individuals, be any increase in the excretion of amino-acid; in cases of hepatic insufficiency increments have been found as high as from two to six times the original values. Using van Slijke’s method, the author found in his case that after the administration of 10 grm. of glycocol the amino-acid-nitrogen secreted in twenty-four hours had been trebled (prior to the taking of glycocol, 650 mgm.; after it, 2,200 mgm.).

3. **Detoxication Function of the Liver.**—When 1 to 2 grm. of camphor are given per os, a considerable quantity should be found in the urine in healthy subjects in the form of campho-glycuronic acid (as much as 75 per cent.). This acid rotates the plane of polarization to the left and can also be estimated quantitatively by Tollens’ method; in his patient, the author found none, or only a trace.

4. **Bile Pigments.**—Bilirubin was not discovered in the urine. The test for bile salts was positive. Urobilin and urobilinogen reactions were positive.

5. **Hepatic insufficiency test of Widal.**—During digestion, protein decomposition products are conveyed to the liver by the venae portae, not only as amino-acids, but also as peptones. The normal liver can retain these peptones completely, but one that is functionally inferior lets them through. In the blood circulation they generate the ‘crise hémoclasique’ of Widal, i.e., decrease in the number of white corpuscles, lowering of blood pressure, modification of the coagulability of the blood, lowering of the refractometric index of the serum.

This reaction of Widal has received considerable attention. With hepatic disorder a leucopenia occurs after ingestion of 200 c.c. of milk, instead of the normal digestion leucocytosis. The figures for the author’s patient are: whites (before food), 5,700; twenty minutes after 200 c.c. of milk, 2,900; forty-five minutes after, 3,200; one and a half hours after, 5,800.

S. A. K. W.


In two cases of epidemic meningitis puncture of the cisterna magna was
performed twenty-four times, and at each puncture, after withdrawal of a suitable amount of cerebrospinal fluid, antimeningocecal serum was injected. Both patients were cured. No untoward effects were observed on any occasion either during or after the operation. It was found that the number of cells in the fluid obtained from the eisterna was always much less than in that obtained from the lumbar theca. In cases of meningitis where frequent punctures are necessary it is convenient to be able to use two routes, and Hardwich considers that subocipital puncture is attended with less discomfort to the patient and that there is less chance of the fluid being contaminated with blood.

J. P. Martin.


The brain of a diabetic subject who succumbed to pneumonia was found to contain multiple gray masses scattered irregularly through the white matter of the left frontal lobe. A section of one of the masses showed the presence of embryonic nerve cells lying in a matrix of delicate fibres. Isolated nerve cells of the pyramidal type could also be found in the normal white matter separating the gray areas. The condition appeared to be due to some abnormal process, acting during the fourth month of fetal life, which to a limited extent had checked the outward migration of neuroblasts in the left side of the forebrain vesicle.

R. M. S.

[107] Parts of central nervous system which tend to exhibit morbid recessive or dominant characters.—Smith Ely Jelliffe. Arch. of Neurol. and Psychiat., 1924, xii, 380.

A critical account of the influence of heredity in nervous and mental disease which will well repay perusal by all interested in a fascinating though somewhat speculative subject.

R. M. S.

SENSORIMOTOR NEUROLOGY.


The symptoms and diagnosis of this condition are discussed. The phenomenon peculiar to pachymeningitis hemorrhagica interna is furnished by an examination of the cerebrospinal fluid, which in this condition is bloody, containing well-formed red blood cells, and shows marked increase of albumin and globulin. Smears are negative and cultures are sterile. A striking feature of the disease is the reappearance of fresh blood in the cerebrospinal fluid following every exacerbation of symptoms, such as headache and convulsions. A case is reported of a heavy beer drinker, male, age fifty-eight years, with no history of trauma. The onset was sudden, with intense headache, vomiting and convulsions. He became stuporous, with severe pains in the muscles, cramp, and paravertebral tenderness. The temperature was 102° F. and the pulse 80. Kernig's sign was present. Four days after