500 per second. Not only is the vibration recognized, but if two forks differ in pitch by a tone or more, their vibrations can be distinguished from each other by the finger. This faculty of distinguishing notes by the finger may even be cultivated, and in deaf children sometimes becomes very acute.

If we admit the existence of a separate vibration sense, many instances of its employment can be given. Katz believes that we rely on it to a very great extent when we tell the nature of a surface or material by moving a finger over it. When we do so vibrations certainly occur, for in many cases a slight noise—rustling or grating in character—is produced. The superiority, too, of the moving finger over the stationary one is known. Touch alone cannot be sufficient, for the differences in elevation on fine materials are much too small to be appreciated by our touch organs. In fact, touch can often be dispensed with; if a short stick be held between the fingers and rubbed on various surfaces, these can be distinguished and their nature can usually be recognized.

The vibration sense frequently also supplements our hearing; e.g., when we become aware of vehicles passing in front of our houses. Like hearing, the vibration sense is responsive to stimuli transmitted from a distance. Probably the animal organism learned to recognize vibratory stimuli long after it had known touch, and long before it knew sound.

To the objection that no central organ for the reception of vibratory stimuli is known, Katz answers that it will be time enough to look for it when we have found separate organs for the recognition and distinction of touch, pressure and temperature. He mentions, incidentally, Frank’s conclusion, that vibratory stimuli are conducted to the brain by special fibres lying in the motor tracts.

J. P. M.

NEUROPATHOLOGY.

[105] The cerebrospinal fluid in adult cases of tuberculous meningitis (Le liquide céphalorachidien dans la méningite tuberculose de l’adulte).
—GEORGES BICKEL. Arch. Suisses de Neur. et de Psychiat., 1923, xii, 269.

This important paper is a review of the examination of the cerebrospinal fluid in eighty-two adolescent and adult cases of tuberculous meningitis. One hundred and eighty-nine specimens of cerebrospinal fluid were carefully examined, and the results are recorded in so concise a manner that it is almost impossible to give a short summary of them.

The appearance of the fluid was clear in 132 specimens; there was a haze in twenty-one, and a turbidity in eight. Turbidity often appeared in later punctures when the earlier fluids had been clear. The pressure was always raised but sometimes fell towards the later stages of the disease. Xanthochromia was present in twenty-two of the fluids, and was associated with the presence of red blood corpuscles in two cases. Tubercle bacilli were found in the majority of fluids (exact number not stated), and were combined with other organisms in three cases. The cells varied from 0 to 1,400 per c.mm., with an average of 230. They were mainly lymphocytes in the great majority.
of fluids, but in 20 per cent. of the cases the earlier fluids showed a pre-
dominance of polymorphonuclear cells; these disappeared towards the end
of the disease, not only in the purely tuberculous cases, but also in those with
a mixed infection.

The protein was estimated in 170 fluids from seventy-six cases by Esbach’s
method. Readings from 0·025 to 0·475 per cent. were obtained, the average
being 0·152 per cent. The protein tended to increase with the ingravescence
of the disease, and often fell slightly during the mild remissions which were
sometimes observed. The only reading above 0·4 per cent. was in a fluid
obtained shortly before death from a case of associated tuberculous and
meningococcal infection. The protein often increased along with the number
of cells, but this was not always the case. Not infrequently the cells were
raised at an early stage of the disease when the protein remained within normal
limits. On the other hand, at a late stage in the disease the cells were
occasionally scanty (18 per c.mm.) and the protein abundant (0·4 per cent.).
In two cases there was no excess of either protein or cells.

The chlorides were examined in thirty-six fluids. In all they were below
normal, but the very low readings which are pathognomonic of the disease
were rare. Thus in only nine cases did they fall below 0·610 per cent.; in
fourteen they were between 0·610 and 0·650, in ten between 0·650 and 0·700,
and in two slightly above 0·700 per cent. The sugar was only estimated in
ten fluids and lay between 0·006 and 0·03 per cent. The percentage of both
chlorides and sugar was found to fall steadily during the course of the disease.

The meningeal permeability to nitrates was examined in five cases and was
always found to be raised, the amount passing into the cerebrospinal fluid
three hours after the ingestion of sodium nitrate (1 grm. for each five stones
of body weight) lying always between 0·004 and 0·0075 per cent., the normal
being 0·001. In one case the permeability was found to increase from 0·0055
to 0·0075 per cent. during the course of the disease.

Unexpected recovery took place in one of the author’s cases in which, at
the height of the disease, tubercle bacilli were shown to be present both
microscopically and by animal injection. At this stage the cerebrospinal
fluid was typical, with 280 cells per c.mm. (60 per cent. lymphocytes), protein
0·3, chlorides 0·595, glucose 0·018, urea 0·02, permeability to nitrates 0·0065
per cent. From this time on the patient and his fluid showed progressive
improvement, the first definitely hopeful sign being the rise in the percentages
of glucose and chlorides, neither of which shows any marked change during
the common slight remissions of the disease. A month from the beginning of
the illness the patient showed a complete clinical recovery, but the cerebro-
spinal fluid was still profoundly altered, containing forty-four cells per c.mm.
(92 per cent. lymphocytes), 0·08 per cent. protein, 0·635 chlorides and 0·037
glucose. The permeability to nitrates, which had remained at the high figure
of 0·0055 per cent., fell rapidly almost to normal. For several months more
the cerebrospinal fluid continued to show about ten cells per c.mm., and a
diminution of chlorides and glucose. As this, so far as the reviewer knows, is
the first non-fatal case of tuberculous meningitis in which careful chemical
examinations of the cerebrospinal fluid have been made, it has been thought
worth while to record them fully.

J. G. GREENFIELD.

In this, his second article under this title (see this JOURNAL, August, 1923), Professor Nishikawa deals with the changes produced in the cerebellum, pons, and medulla by tumours in the cerebellopontine angle.

In many cases there was a great increase in the size and number of the blood vessels in the whole field around the tumour; sometimes the vessels showed changes in their walls—weakening of the intima, hyaline degeneration or simple thickening of the wall; thrombosis was frequently found, and at times the occlusion of a vessel in this way seemed to have been the cause of necrosis of brain tissue in the area of distribution of the vessel.

In the cerebellum, besides these changes in the vessels, there were in some cases areas of hæmorrhage and blood spaces in communication with arteries; in other cases the tumour itself had invaded the cerebellum, either massively or by tentacles growing along the vessel sheaths; in other cases, again, areas of softening were found, and slighter degenerative changes due to pressure.

In the pons most cases presented a slight œdema, but the fibre tracts and nuclei in the deeper parts seemed to have escaped injury. But in the pontine peduncle of the cerebellum, situated in proximity to the tumour, there were areas of partial degeneration, the myelin sheaths apparently being destroyed while the axis cylinders escaped; in other cases the peduncle had been attacked by the growth and seemed to have been the path of invasion of the cerebellum.

In the medulla the involvement was much more serious and extensive. The whole area corresponding to the distribution of the posterior inferior cerebellar artery had in one case undergone softening; this was a case in which operative interference had been quickly fatal; the softened area included the nucleus of the vagus nerve.

Nishikawa draws from his studies three practical conclusions: (a) that death after operation for cerebellopontine tumour may be due to the peculiar vascular conditions existing at and around the site of operation; (b) that as some of these tumours invade the cerebellum extensively (and are probably diagnosed earlier on that account) it must in some cases be considered whether the operation should not be planned as for a cerebellar tumour; (c) that no operation should be undertaken in any case in which there are definite signs of medullary lesion. It is recognized, however, that the question whether certain symptoms are produced by affection of the medullary nuclei themselves or by that of the nerve roots which arise from them is usually extremely difficult to decide.

J. P. MARTIN.

[107] Syphilis of the small cerebral blood vessels (La syphilis des petits vaisseaux du cerveau).—URECHIA and ELEKES. L’Encéphale, 1923, xviii, 240.

The authors detail a case of chronic syphilitic disease of the brain corresponding to the type described by Nissl and by Alzheimer as ‘diffuse cerebral
syphilis.’ In this condition the brain is usually reduced in weight and the meninges are thickened, but the histological changes differ in many respects from those of general paralysis. There is little perivascular round-celled infiltration, but, on the other hand, the walls of the smaller vessels show proliferation of both intima and adventitia, often with disappearance of the media, and fragmentation or proliferation of the elastic lamina. The nerve cells present chronic alterations; sometimes neuronophagy is intense. The neuroglial cells are swollen, and have large nuclei, but new formation of neuroglial fibres is scanty, in contradistinction to the condition found in general paralysis where the neuroglial overgrowth is largely fibrous in character. In the authors’ case there was also calcareous infiltration in the lenticular nuclei, affecting chiefly the adventitia of the vessels but sometimes infiltrating all the coats and even penetrating into the interior of the vessel to form ‘actual stalactites.’ In addition, small round dots of calcium salts were found scattered through the tissues and grouped in the neighbourhood of the capillaries.

The nature and pathogenesis of this calcareous infiltration is discussed, and a short but exhaustive review of the literature dealing with the subject is given. The authors take the view that the lime is in the form of a colloidal combination with iron and fatty acids, and that its deposition is due to diminished alkalinity of the blood.

That the condition was of syphilitic origin in this case was proved by the presence of syphilitic aortitis and nephritis, although the Wassermann reaction was constantly negative both in blood and cerebrospinal fluid.

J. G. Greenfield.


It is no longer held that syphilis is a hereditary disease in the strict sense of the term; either the ovum becomes infected from the father at the time of its fertilization—in which case it almost certainly dies long before term—or the fœetus becomes infected subsequently from the mother.

Colles’ law that the mother may escape infection and yet bear a syphilitic child cannot be sustained in the light of modern investigation; for Trinchese found spirochætes in plenty in the decidual (and therefore maternal) part of the placenta, even in cases where the mother’s blood gave a negative Wassermann reaction. While this shows that the mother does not escape, it does seem, nevertheless, that her infection may be of a peculiarly mild type; Salomon suggests as an explanation of this that pregnancy, in much the same way as the modern non-specific pyrexial treatment, may have a favourable influence on the course of syphilis.

Profeta’s law, that a syphilitic mother may bear a non-syphilitic child, became untenable when the Wassermann reaction was introduced; though infection may not be apparent in the child it can be detected in his blood; it is certain, too, that no immunity is conferred upon the child by the mother and that no immune bodies pass to him from her blood.
ABSTRACTS

The infection of the child may occur (1) at any time during gestation, or (2) during parturition, either (a) by the placental route or (b) cutaneously.

Salomon concludes that though a man with a positive reaction in his blood may beget healthy children, it is not to be expected that a woman with a positive reaction in her blood will bear healthy children, and she should not therefore be allowed to marry.

J. P. M.


There is a variety of causes for the formation of lacunae in cerebral arteriosclerosis. The type described by Marie seems to be due to a perivascular proliferation which corrodes the surrounding nervous tissue. This sometimes leads to rarefaction but more often to a proliferation of neuroglia in the vicinity; the connective tissue tends to break down in the centre, and this brings about the formation of lacunae. Small circumscribed lacunae such as are seen in chronic subcortical encephalitis are due to reabsorption of small perivascular hemorrhages and to the softening of miliary thrombi. The 'cribriform' condition described in the cerebral white matter of general paralytics, senile dementias and also epileptics, depends on interference with perivascular lymphatics and the dilatation of lymphatic spaces round the small arteries.

'L'état vermoula' described in senile dementia, consisting of cortical circumscribed areas of softening or cavities surrounded by a dense layer of neuroglia, is due to necrosis following on occlusion of blood capillaries. There is no demonstrable difference in the clinical picture accompanying these various forms of degenerative change.

R. G. Gordon.

[110] Lesions caused by histamine in the nervous centres of the rabbit (Lesioni provocate dall'istammina nei centri nervosi del coniglio).—V. M. Buscaino. Riv. di Patol. nerv. e ment., 1923, xxvii, 641.

The author believes that the pathogenesis of certain morbid processes such as those of dementia praecox, amentia, the chronic form of encephalitis lethargica, Parkinson's disease, progressive muscular atrophy, and perhaps Wilson's disease, consists of an intoxication by the amine compounds.

The effects of intravenous and subcutaneous injections of histamine into animals are described. They consist in profound disturbances of the vegetative nervous system, such as stimulation of smooth muscle fibre, salivation, asthma, lowering of blood pressure, etc. The author conducted experiments by injecting rabbits with histamine and examining their nervous systems, and found areas of degeneration similar in character and distribution to those met with in dementia praecox, both in the glial tissue of the central nervous system and in the liver. A dark precipitate was obtained in the urine treated with silver nitrate. Similar conditions have been described in the other disease processes mentioned above, and it is significant that the lesions in the rabbits experimented on were confined to the basal ganglia and the mesencephalon. He concludes that these degenerative diseases are due to the presence in the circulation of an abnormal basic substance, probably histamine.

R. G. Gordon.

This is a long article in which the detailed description of the pathological findings in the brain in a case of hypertrophic tuberous sclerosis is followed by a discussion of the etiology of the abnormalities observed.

The case was that of an infant aged fourteen months. The brain showed to the naked eye typical 'tubera' on the surface in the frontal and temporal lobes of both sides and in the right parietal lobe. On section of these nodules it was seen that they involved both the grey and the white matter, and that the grey matter in them was thinner than over the more normal parts of the brain.

On microscopic examination of the nodules, the grey matter involved in them was found to be a kind of miscarried cortex, i.e., the usual cortical elements were present, but in arrangement and in form they showed considerable disorder. As regards arrangement, it was impossible to make out definite layers of cortical cells. As regards form, both size and shape were very varied; the cells were multipolar, bipolar and unipolar; giant cells, some of them of enormous dimensions, were present in great numbers; many of these were definitely ganglion cells, others approached closely to the glial type; Pollak satisfied himself, however, that these latter were not true glial elements, and that they were to be reckoned with the ganglion cells.

In the white portion of the nodules, unusually small glial cells in great multitude dominated the microscopical picture, and there was also a great excess of glial fibres.

Passing from the nodules to the apparently normal parts of the brain, Pollak found giant ganglion cells in several parts of the cortex, and he also discovered great numbers of ganglion cells, some of large size, some of normal size, in the white matter in the internal capsule.

To explain his findings the author suggests that two separate pathological processes have been at work during development. The first he supposes to be some influence which at an early stage acted on the neuroblasts and spongioblasts in such a way as to pervert their properties of growth and to interfere with their biotaxis. This would explain the variations in form and size of the ganglion cells, the incomplete differentiation of some of them, and their presence in abnormal situations. The second process he takes to have been a focal reaction of the glia around the more abnormal cells—the usual glial reaction of the nervous system to damage of almost any kind; but owing to the perverting action of the earlier factor the glia is not true to type, and its cells show almost as much variation as the true nervous elements.

J. P. Martin.


Since the researches of Chevreul and Liebig, in 1847, creatin has been recognized as a constituent of muscle tissue and as the forerunner of the creatinin which is constantly present in healthy urine. The dehydration of creatin to


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creatinin was generally supposed to be brought about by ferments, but in 1920 Hahn and Barkan showed that the change occurred spontaneously in an acid medium, if the degree of acidity was about the same as that normally present in the muscles of the body. The daily output of creatinin for a healthy man taking a creatin-free diet is about 1 grm., and if the diet be not only creatin-free, but nitrogen-free, the creatinin nitrogen amounts to between 18 per cent. and 26 per cent. of the total nitrogen output.

Since 1883 it has been known that in cases of muscular dystrophy the creatinin output is reduced. In 1909 Levine and Kristeller found that in muscular dystrophy and in conditions of muscular wasting generally, not only was the output of creatinin reduced, but creatin itself appeared in the urine. These findings were confirmed by other workers, both in this country and in Germany.

Nedelmann has now investigated the relation of the creatinin output to the total protein output in a case of muscular dystrophy. The patient was placed on a nitrogen-free diet and estimations were made daily from the sixth till the tenth day; the average daily excretion of creatin was just over 0.41 grm. (41 per cent. of the normal), and the creatinin nitrogen was only 9.9 per cent. of the total daily output, i.e., half the normal ratio.

J. P. M.


In the view of the author, supported by full evidence, Landry's paralysis cannot be considered a clinical, pathological, or etiological entity. Yet, regarded as a syndrome, it is definite enough—an acute ascending (or descending) flacid motor palsy, not always fatal, with occasional amyotrophy, occasional involvement of sphincters and vegetative centres, very occasional sensory changes. The unity behind this diversity is, in the author's opinion, to be sought in the preliminary state of the nervous system of the individual concerned, whereby resistance is lowered and reaction is merely passive.

In an interesting and well-considered discussion a search is made for the 'predisposing coefficient' of the syndrome. This consists in a state of 'toxavidity,' a 'sensitisation' of the neuraxis as the result of persistent toxic infection or the presence of foreign substances. The source of this preliminary sensitisation may be extrinsic or intrinsic, the latter, autointoxicative, group being exemplified by a large number of conditions (renal and alimentary toxemias, polyneuritis gravidarum, idiopathic or recurrent polyneuritis, polyneuritis neurasthenica, polyneuritis ambulatoria, the polyneuritis of avitaminosis, etc.) in which it is clear that toxic action on the nervous system is taking place from some nidus in another somatic system. Thus prepared, the nervous system is prone to succumb to an acute invasion whatever the actual morbid agent.

Among these predisposing coefficients is to be numbered the comparatively rare condition of haematoporphryia, or porphyrysm, as it may shortly be called, and Grünewald gives a full description of a remarkable case of this kind, ending with the characteristic clinical picture of acute Landry's
paralysis. As in a previous paper of the same writer dealing with the same subject (see this JOURNAL, May, 1923, p. 54), it is to be regretted that the title of the communication conveys no information as to its interesting contents.

Summarized, the thesis is that Landry’s paralysis is a special biological expression of differing morbid processes, of an unfavourable nature, in which behind the clinical symptoms lies a biological preliminary in the form of toxic action on a nervous system which reacts anaphylactically (allergically) by toxic over-sensitisation.

S. A. K. W.

[114] Is the albumin-content of the cerebrospinal fluid different at different levels? (Bestehen Unterschiede in Eiweissgehalt des Liquor cerebrospinalis in verschiedenen Hohen?)._JACOBI. Münch. med. Woeh., 1923, lxx, 670.

It has been shown almost beyond doubt that the cell content of the cerebrospinal fluid is different at different levels, and that the cerebrospinal fluid as a whole is not to be judged in this respect by the result of a single examination. The question then arises whether similar variations occur in the albumin content. That such is the case has been suggested by the findings of Walter and others who carried out albumin estimations on consecutive samples drawn off at the same puncture. Eskucheich, Matzdorff and Schonfeld, however, have not been able to discover such differences.

The author of this paper attacked the question armed with a new method of examination: by means of the ‘interferometer,’ which determines optically the strengths of solutions, he examined, in each of his cases, five consecutive specimens of fluid taken at the same puncture. In this way he studied thirty fluids. His findings are in keeping with those of Walter, and tend to show that the albumin-content does vary slightly at different levels, and is usually highest in the first sample of fluid drawn off.

J. P. M.

SENSORIMOTOR NEUROLOGY.


An old man of eighty-two suffered from a stroke, without loss of consciousness, which was followed on the fourth day by the appearance of pronounced intention-tremor of the right arm, the leg to a less extent, and of the upper part of the trunk, but excluding the head. At an autopsy two years later hemorrhagic softening of the right nucleus dentatus of the cerebellum was found, with secondary degeneration of the superior cerebellar peduncle. The nucleus fastigii was intact, but the nuclei emboliformis and globosus were implicated. The right middle peduncle was also degenerated, and the contralateral inferior olive and olivo-cerebellar tract in the medulla.

Leiri thinks the intention-tremor of cerebellar disease is due to an attempt on the part of the cortex to correct exaggerated, hypermetric movement the result of the cerebellar lesion. The varying degree of representation in the