The difficulties enshrouding the pathological physiology of cerebral concussion are to some extent dispelled by this long and elaborate paper, based on the results of innumerable experiments, mainly with dogs, and correlated with experiences of clinical cases of war and other origin. A full account is given of the technique employed, the animals being narcotised and curarised, the effects of blows on the head being observed by appropriate apparatus comprising the registration of the general blood pressure, the volume of the brain, the venous return from the brain, the volume of the abdominal viscera, etc. Some fifty-eight experiments were made on twenty-three animals. The authors’ first conclusion is that an immediate result of a severe blow on the head is usually a sudden increase, less often a sudden decrease, in the amount of the circulating cerebral blood, produced by action on the heart and vessel centres in the medulla oblongata, via the cerebral vasomotor apparatus. This immediate effect is rapidly followed by a more or less prolonged state of reduction in brain-volume and relative emptiness of the cerebral vascular system, in all probability due to diminution of vasomotor tonus. Since the vessels, in this state, will nevertheless respond to the constricting action of adrenalin, it seems clear that the cause of the loss of vasomotor tone is central, i.e., by action on vasomotor centres in the medulla, and so on vasomotor nerves, which can be shown to be in a state of reduced excitability.

The explanation of the sudden loss of consciousness in severe cerebral concussion or commotion is fully examined. It cannot in its entirety be put down to the vascular change as specified above; this at the most is an accompanying but not a causal phenomenon. The authors adduce interesting evidence which suggests that the coma is the immediate consequence of action on cerebral neural elements themselves, and that the site of this action is not solely cortical (the condition of decerebrate rigidity is very different from that of coma), nor medullary, nor mesencephalic; action must implicate cortex, midbrain, and medulla more or less simultaneously and completely to produce the clinical picture of coma from concussion.

S. A. K. W.

A useful précis of a large number of reported cases of Landry's paralysis from about 1905 is presented to show the extreme pathological indefiniteness of the condition, precise enough though it appears to be clinically—an acute, often ascending, flaccid palsy of the limbs and trunk, without sensory disorder and usually without involvement of the sphincter reflexes. The author's own case is one of the remarkable group associated with hemato-porphyrinuria, and is worked out with the greatest minuteness; its documentary value is considerable. Such cases have been classed with polyneuritis (by Harris and others), and it is perhaps matter for regret that the title of this paper does not indicate the nature of the rare case therein detailed.

Briefly, the changes in the peripheral nerves were those of 'névrite segmentaire périaxile,' in combination with ordinary secondary or Wallerian degeneration. The former is characterised by local myelin disintegration with overgrowth of the protoplasm and nuclear elaboration of the cells of the sheath of Schwann. In the central nervous system were found acute degeneration of the cells of the lumbar and dorsal cord, regressive changes, glial proliferation, Wallerian degeneration, and transudative alterations in the vicinity of vessels. Similar changes were present in the cervical cord, and to a less extent in the brain. A long discussion on the nature of these alterations is concluded in the decision that they represent acute toxi-degenerative and not inflammatory processes.

Landry's paralysis is a syndrome encountered clinically in cases of poliomyelitis, myelitis, polyneuritis, and epidemic encephalitis. Pathologically it ranges from a negative or almost negative finding to the fully developed changes of a meningo-encephalo-myelo-neuritis. The author accordingly is convinced that etiologically and pathologically the expression 'Landry's paralysis' is meaningless.

S. A. K. W.


This interesting paper is based on a critical examination of the description (supplemented in one instance by the examination of sections loaned to the author) by various writers of the pathological appearances in cases of hypertrophic neuritis which they have published. It is a somewhat bold procedure to challenge the interpretations of the writers themselves when the challenger has apparently no personal material to support his conclusions, but Bielschowsky's position as a neuropathologist is pre-eminent and his analysis of the subject peculiarly instructive.

Briefly, his thesis is that he is surprised to find that no one, it seems, has thought of correlating the findings in familial hypertrophic 'interstitial' neuritis with those of Recklinghausen's disease, especially with that form known after Verocay's researches as 'neurinomatosi.' Examination of the
former shows undoubtedly, according to Bielschowsky, that the hypertrophy is due to the appearance of concentric rings round the axones; they look as if they were hypertrophied myelin sheaths, whereas in reality they are made up of an almost homogeneous or extremely fine granular substance in whose periphery the Schwann cells are embedded. In this plasma-like ground-substance minute fibrils can be differentiated, and these constitute the ectodermic 'nerve-fibre-tumour' appearance characteristic of neurinoma. The process appears to commence by proliferation of the Schwann nuclei in given Ranvier segments of the nerve, with simultaneous increase in volume and thickening of the cytoplasm of the Schwann cells in the same segments. Hence concentric lamellæ, producing an 'onion-skin' appearance, eventually develop, being laid down, presumably by some as yet not recognized morbid biochemical process, within the Schwann sheaths, a condition found in no other variety of neuritis. The process is not inflammatory but blastomatous, and is entirely analogous to what is found in so-called 'polycentric (peritubular) neurinomatosis.' In a word, the tendency to blastomatous proliferation within the Schwann sheaths over the whole extent of the nerve is identical with the limited, local, condition found in the variety of Recklinghausen's disease known as neurinomatosis. It is of interest to note the more or less constant presence of minute regenerating nerve-fibrils (quite distinct from the neurinomatous fibrils) within the Schwann sheaths, often in enormous numbers; as a rule, they, too, eventually degenerate, as do the original axones of the nerve, from continuation of the morbid process, but their occurrence may explain the often relatively well-preserved function of hypertrophied peripheral nerves.

The term 'interstitial,' therefore, is undesirable, inasmuch as by far the larger part of the hypertrophy is of ectodermic and not mesodermic origin; the part played by the connective tissues is quite secondary. The hypertrophy of familial hypertrophic neuritis is a universal peritubular neurinomatosis, continuous from periphery to roots.

S. A. K. W.


The 'paregoric reaction,' which is closely allied to the colloidal benzoin reaction, consists simply in adding a few drops of 'paregoric' to a dilution of cerebrospinal fluid in distilled water. In a technique previously published, the author used five tubes containing 1 c.c. of dilutions of \( \frac{1}{4}, \frac{1}{4}, \frac{1}{8}, \frac{1}{16} \) and \( \frac{1}{32} \) of C.S. fluid in distilled water, and added 0.2 c.c. of paregoric to each tube. Normal fluids gave no precipitation of the paregoric in the first two and last of these dilutions, with varying degrees of precipitation in the third and fourth tubes. The fluids of general paralytics, on the other hand, gave complete or partial precipitation in the first four tubes.

Targowla has now altered his technique and uses only one tube, in which are put 0.25 c.c. of distilled water, 0.75 c.c. of the C.S. fluid to be tested, and 0.3 c.c. of paregoric; a control tube receives 1 c.c. of distilled water and
0.3 c.c. of paregoric: the tubes are then shaken well and are read at the end of twelve to twenty-four hours.

In sixty-three out of sixty-five cases of general paralysis the reaction thus performed was positive, i.e., precipitation occurred in the tube containing the fluid to be tested. The two negative cases were tested during long remissions of symptoms. We are told that the reaction was always negative with the cerebrospinal fluid of non-paretic patients, but no figures are given of the number or the nature of the cases examined.

J. G. Greenfield.


The following groups of pathological alterations are recognizable in the choroid plexuses: (1) Alterations having their origin in the interstitial and perivascular mesodermal tissue (proliferation, cystic degeneration, vascular processes, etc.) capable of bringing about secondary transformations of ependymal cells; (2) chronic and acute alterations arising in the glandular ependymal tissue, causing primary destruction of the protoplasm, followed by that of the ependymal cells, and by sclerosis and atrophy *en masse* of complete groups of these cells. These alterations are generally accompanied by atrophic processes in the ventricular ependyma and the subependymal substance, and also by the penetration of pathological products into the nervous parenchyma; (3) mixed mesodermic and ectodermic alterations.

The mesodermic type of alteration of the choroid plexuses is less noxious and may remain latent longer. It is, nevertheless, probable that if it goes beyond certain limits it may give rise to conditions of somnolence, stupor and mental confusion. On the other hand, the ectodermal (or mixed) type of alteration is more dangerous for the intimate physiology of the nervous processes. In cases where alterations of the glandular and ventricular ependyma are advanced and diffuse (particularly those in the fourth ventricle and in the inferior horn) they are accompanied by serious mental trouble, particularly affecting the emotional life, and by acute as well as chronic states of delirium with hallucinations, obsessions, negativism, etc. That the lesions of the plexus cause the mental symptoms is unproved and unlikely, but the importance of the influence of such lesions of the plexus on the cortex is unquestionable.

R. G. Gordon.


1. Chronic Chorea.—A useful summary of recently published cases of Huntington’s chorea, examined by modern methods by various workers, is followed by an account of the findings in four personal cases. Special attention is directed to the fact that the syndrome of chronic chorea may be unaccompanied clinically by mental symptoms and pathologically by cortical changes. The main features of Bielschowsky’s cases were as follows: little recognizable
change in the cortex; pronounced loss of cells in putamen and caudate, such cells as remained being chiefly of the large type and exhibiting much pigmen-
tary degeneration; great overgrowth of fibre-forming glial cells every-
where in the same ganglia; slight tendency to cystic or porous degeneration
in the tissues, with degenerated blood-vessels in relation; products of degener-
ation not at all obvious, with the exception of fatty inclusions in the cytoplas-
m of the fibre-forming glial cells; notable vascular changes, mostly thickening
of capillaries (capillary fibrosis), the more obvious, perhaps, because of the
general shrinkage of the putamen-caudate, and also some capillary new-
formation and connective-tissue ‘bridges’ between neighbouring capillaries;
a characteristic status fibrosus of the ganglion, produced by loss of myelinated
fibres (intrastratal and striofugal) and assembling of those remaining into
thicker strands owing to the shrinkage of the ganglion—hence a general denser
and darker coloration with myelin stains; similar but slighter alterations in
the globus pallidus; corpora arenacea and corpora amyloidea in both parts of
the corpus striatum.

There is no evidence of any selective action of the morbid process on the
small-cell systems of the ganglion, as Ramsay Hunt has supposed. Biels-
chowsky claims that in chorea chronica this histological picture is “clear-
cut and constant,” yet he is compelled to allow that the status fibrosus occurs
in cases which do not belong clinically to chronic chorea; it is seen, e.g., as
a sequela in some cases of infantile encephalitis, and he cites a personal case
where this pathological condition was found and in which neither athetosis
nor chorea was present clinically.

2. Progressive Rigidity.—A case is given in great pathological detail in
which a boy of six began to suffer from generalized choreiform movements;
these some years later gradually ceased, to be replaced by progressive rigidity
of limbs and trunk, and in fact of the whole of the skeletal musculature.
Pathologically, diffuse changes were found mainly in the third cortical layer;
the putamen-caudate was in a condition of status fibrosus, while the globus
pallidus was notably degenerated and shrunken, much more so than in
Huntington’s chorea; pronounced chronic cell-changes were present also in
the corpus Luysii, substantia nigra, and nucleus ruber. The argument is
that the involvement of the pallidal and subpallidal centres is responsible
for the progressive rigidity, masking the choreiform movements. It is held
that in cases of Huntington’s chorea in which the globus pallidus is implicated
the degree of involvement of the latter has not been sufficient for the develop-
ment of rigidity, but the evidence offered is not convincing. Bielschowsky
argues that in decerebrate rigidity the putting out of action of the ‘tween-
and mid-brain centres belonging to the pallidal system is a factor in the pro-
duction of the rigidity, but the selective nature of the latter in decerebrate
circumstances is different from the generalized hypertonus of striatal cases.

Reference is made to the Ramsay Hunt hypothesis in connection with
juvenile paralysis agitans, and Bielschowsky criticizes it adversely.

3. Paralysis Agitans.—The description given is based on the investiga-
tion of six typical cases of the disease. The chief alterations were: constant
loss of cells (both types) in putamen-caudate, and in globus pallidus; chronic
fatty and lipoid degeneration of the cells remaining; fatty points surrounding
the numerous glial nuclei and seen in the nuclei of the walls of the smaller
blood-vessels; products of degeneration in adventitial cells and in occasional
compound granular corpuscles; diminution and loss of myelinated fibres,
more notable in globus pallidus than in putamen-caudate, and also in the ansa
lenticularis and in Forel's field; angiosclerosis of the larger blood-vessels,
and capillary fibrosis, both as a rule prominent; in association with these
changes in the ground-substance of the ganglion, a sort of rarefaction analo-
gous to a status prerubratus or eribratus, and histological evidence of venous
stasis and interference with lymph percolation through tissue spaces—these
changes more obvious in the putamen-caudate than in the globus pallidus;
abundant products of defective metabolism and degeneration, a question
standing much in need of further histological and chemical research.

The author considers it a mistake to regard the pathological process in
paralysis agitans as limited to the corpora striata, since he has seen degenera-
tive changes also in cortex, optic thalamus, corpus Luysii, substantia nigra,
and in various areas in pons and medulla. He confesses his inability to deduce
any precise explanation of the rigidity and tremor from a consideration of his
pathological findings, though he thinks it justifiable to hold that the striatal
changes form the anatomical basis of the clinical syndrome.

4. Pseudosclerosis and Wilson's Disease.—In his discussion on these
diseases and on their possible interrelation, Bielschowsky points out clearly,
as he has done before, the essential dissimilarity between the chronic blas-
toma-like glial hypertrophy of the former and the acute or subacute parenchym-
atous and interstitial degeneration of the latter; he says in so many words
that 'in their original forms the two diseases have nothing in common.' He
discusses cases published subsequent to Wilson's papers in which apparently
a combination of the two distinct processes has been noted (Spielmeyer,
Stöcker), and shows conclusively that there can be no specificity attached
to the large glial cell-forms found by Alzheimer and others in pseudosclerosis,
since they have been seen in a number of other conditions. In spite of the
work of Spielmeyer the histopathological differences between the two diseases
are still unbridged. Bielschowsky gives the details of a personal case of
Wilson's disease in which, notwithstanding his careful search, he has com-
pletely failed to find any evidence of the presence of the giant glial cells as
seen in some cases (not all) of pseudosclerosis.

The paper is of great importance and value, yet it serves to show how far
the morbid histology of these interesting striatal diseases is from shedding
light on the physiology of their symptoms.

S. A. K. W.

[7] The pathological anatomy of paralysis agitans (Zur pathologischen
Anatomic der Paralysis agitans).—FÜNFGELD. Zeit. f. d. g. Neur. u.
Psychiat., 1923, lxxxi, 187.

The case described is that of a man of sixty-seven, the duration of whose
symptoms, in every way characteristic of the disease, did not extend beyond
fourteen months; a complication was present in the form of a degree of
senile dementia.
Cortex.—Marked shrinkage, fatty degeneration, and pigmentary atrophy of many of the cells of the third, fifth, and sixth cortical layers, especially in the frontal region and to a less extent in the occipital.

Basal Ganglia.—In putamen and caudate, which were not obviously reduced in size, moderate atrophy of the small cells and notable degeneration of the large cells, of the usual chronic type, with similar finding in the globus pallidus, especially dorsally and externally. The cells were shrunken, stained more darkly, had lost their normal contour, their processes were reduced and their nuclei pale and poor in chromatin. Glial nuclei were not increased in numbers. Lipid degeneration was readily seen in the cytoplasm of the small cells, in little collections round the glial nuclei, and in the lymphatic sheaths of the vessels. No obvious reduction in myelinated fibres, no obvious increase of glial elements. Pigmentary degeneration of many of the glial cells, enlargement of perivascular spaces, and changes in vessel walls (hyaline and other forms of degeneration). No particular alteration in the ansa lenticularis. Marked reduction and degeneration of the cells of the nucleus substantiae innominata; atrophy of the nucleus periventricularis. A moderate degree of pigmentary atrophy in the optic thalamus (chiefly ventral).

Regio Subthalamica, etc.—Reduction in size of the corpus Luysii, with degeneration of its cells. Marked changes in the substantia nigra, and in the posterior third of the nucleus ruber. Similar degenerative processes in the nucleus dentatus of the cerebellum, the dorsal nucleus of the vagus, and of the cells in relation to the mesencephalic root of the fifth.

The author ascribes considerable importance to degenerative changes in the ground-substance of the basal ganglia, the result possibly of lymph stasis in the tissue synctium. He passes no comment on the interpretation of his findings, except to suggest that the changes in the corpus striatum are analogous to those of the cortex in senile dementia. The process at work in paralysis agitans, therefore, might be considered, in his view, a senile morbid process, with its incidence on the basal ganglia.

S. A. K. W.


Two cases of paralysis agitans, two of Huntington’s chorea, and one of congenital double athetosis were examined, and it was found that the blood creatinin values were very definitely below the normal minimum, affording an indication of the possibility of concomitant alteration in muscle metabolism.

R. G. Gordon.


There can be no doubt that local freezing of nerve trunks is the best procedure to ensure adequate interruption of function, for a sufficiently long period, without reduction of their power of subsequent regeneration. Analysis of the exact histological changes has been undertaken by the authors, who
experienced on guinea-pigs and dogs by exposing various peripheral nerves and freezing them with ethyl chloride for varying periods from one minute upwards. The first change, recognizable in twenty-four hours, is an action on the blood-vessels of the nerve; neural degeneration and regeneration can be seen by about the third day. The former consists in hyperemia of the capillaries and smaller veins, with diapedesis of both whites and reds; within three days circulation is restored and the extravasated blood absorbed. The latter shows itself in breaking up of the myelin sheaths, fragmentation of the axons, and alteration of the cells of the sheath of Schwann into scavenger cells. The process of regeneration begins forthwith and is recognizable by the appearance of minute fibrillary processes spreading peripherally from the proximal ends of the fibrils of the injured axones, and by collateral branching somewhat more centrally than the extremities. There is to be found, after a fortnight, continued evidence of seavenging, in which the mesodermal elements of the endo- and peri-neurium take a prominent share, the reasons being that the extent of the reaction in the nerve passes considerably beyond the section actually frozen, while the rapidity of the degeneration is greater than in ordinary cases of neuritis or of Wallerian degeneration. By the fifty-sixth day the authors have obtained histological proof of complete regeneration by means of new fibres and myelin sheaths.

From the practical viewpoint of the treatment of neuralgia, etc., it is concluded that freezing is altogether preferable to alcohol injection where mixed nerves are concerned; alcohol produces a coagulation-necrosis which is sometimes never recovered from, and has the further disadvantage of tending to cause an intraneural neuroma-formation which may prove a complete barrier to restoration of function.

S. A. K. W.

SENSORIMOTOR NEUROLOGY.


Krabbe has observed two cases of tuberose sclerosis of the brain, and one case of hydrocephalus associated with precocious puberty. Of the former, one case ended fatally at the age of four years, and the diagnosis of tuberose sclerosis of the brain with some degree of microcephaly was established post-mortem. In the other case the patient, a boy of fourteen, was alive but progressively imbecile, and presented the typical sebaceous adenoma of the face. The case of hydrocephalus was only remarkable in that menstruation had commenced at the age of eight years; hydrocephalus, which originated in infancy, appears to have followed a mild attack of epidemic cerebrospinal meningitis. The patient died at the age of forty-four of pneumonia, complicated by glycosuria. Two cases of epilepsy are also described in which the onset of puberty was unusually early. Krabbe refers to the nine recorded cases in which a pinacl tumour was associated with precocious puberty, and considers that the evidence they present is quite insufficient to prove any