The hypothalamic centres of the autonomic system were the first objects to study, but more recently evidence has been adduced to show that centres in the cortex, especially in the motor cortex, were concerned with autonomic activity. To investigate this the conduction of cortical impulses to vegetative organs was studied in 35 cats.

In a first series of experiments 'extrapyramidal' centrifugal fibres arising from the hypothalamus were severed. In a second series of experiments a transverse section of both pyramidal tracts was made. After the lesion on either of these systems had been performed, the effects of stimulation of the motor cortex and the frontal lobe were noted on the pupil, the blood vessels, the sweat glands, and the urinary bladder.

In both groups of experiments the cortical stimulation elicited reactions of the above-mentioned organs. Severance of the hypothalamic pathways impaired most the conduction of corticofugal impulses to the dilator of the pupil, least the conduction to the bladder. It is concluded that there exists a double (pyramidal and extrapyramidal) conduction of corticofugal impulses to the autonomic centres in the cord.

R. G. G.

In view of the fact that a continuous supply of blood to the brain is essential it is somewhat surprising that the number of capillaries in the grey matter of the brain is meagre compared with that of muscle.

The findings of the present study tend to show that the metabolic activity of the brain is safeguarded by a speed of blood flow which is more constant than in the extremities. Within limits, therefore, a constant supply of oxygen is more important to the brain than a rich supply.

R. M. S.

There are at least three possible causes of disturbance of behaviour after an injury to the cerebral cortex.

First, the functional organization may be disturbed by direct damage
to the nerve elements involved. Second, the destruction of cells in one region may result in functional disturbance in cells in other regions of diaschisis. Third, the metabolism of adjacent or even remote parts may be disordered by alteration of the vascular supply. Vascular supply may not coincide with the region of direct damage to nerve-cells, but, in general, vascular deficiency in a cortical field always implies functional abnormality although a functionally abnormal area does not necessarily mean the presence of vascular disturbance. In the case of a lesion with actual removal of tissue, there is always a zone bordering the site of the operation in which both cellular and vascular changes are apparent. The present study was made in an attempt to determine the relative contents of the two disturbances after a lesion is inflicted on a cortical field and to discover whether vascular changes sufficient to account for functional disturbances can be demonstrated at greater distance from the site of operation.

Cortical lesions in nine rats studied by the injection technique yielded the following results. The vascularity of such regions was apparently increased. This was evidenced by one or more of the following changes: a greater number of blood vessels, a larger diameter of the feeding vessels and richer capillary anastomoses. This was true irrespective of the topographical or functional locality of the lesion, its size, shape, the depth reached, the length of time from operation and the manner of inflicting the injury.

R. M. S.


The 'chronic' decorticated dog is able to distinguish light from darkness, to appreciate warmth and tactile sensation, and to hear. The ability to hear extends to distinction between sounds which are qualitatively different and extends over a fairly large range of intensity, although the threshold is high.

The decorticated animal exhibits physiological changes referable to vegetative function and not explainable in alteration of the somatic motor and sensory functions.

The decorticated animal does not exhibit incessant activity, but shows an inability to initiate or inhibit movement suddenly. The distribution of tonus is abnormal, and posture is disturbed.

The decorticated animal exhibits a low sugar content of the blood, with an altered tolerance for dextrose, a high calcium content, increased sensitivity to the action of drugs and disturbed gastrointestinal function. There is an abnormal blood picture with decrease in the erythrocyte count. The pulse pressure is small.

Evidence is presented which leads to the conclusion that both the
sympathetic and the parasympathetic systems are normally inhibited by the
cortex and that removal of the cortex releases both of these systems.

R. M. S.

[65] Hyperthermia due to lesions in the hypothalamus.—B. J. ALPERS.
Arch. of Neurol. and Psychiat., 1936, 35, 30.

Serial sections of the hypothalamus were studied in two cases of suprasellar
cyst in patients who died with hyperthermia in order to determine what areas
are injured in this type of disorder of temperature regulation.

The nature of the hyperthermia, with its steady upward trend, death
within 48 hours and no appreciable drop, was characteristic of the temperature
reaction following manipulation around the region of the pituitary gland in
fatal cases.

The point of greatest interest from the standpoint of localization was the
position of the pathological changes in the region of the substantia grisea
of the third ventricle, with destruction of the adjacent part of the tuber
cinereum, and in the first case, of a small portion of the nucleus tuberomammillaris.
All other hypothalamic nuclei escaped unharmed. The destroyed region in
the cases corresponded closely with the areas involved in cats exhibiting
loss of temperature regulation. In these animals the infundibular nuclei of
Winkler or the anterior hypothalamic nuclei of Rioch were found to
be affected chiefly. While it is impossible to compare, area for area, the hypo-
thalamic nuclei of the cat and of man because of definite structural differences
in this region, it is possible, nevertheless, to indicate that the regions disturbed
in the cases of hyperthermia reported by the author corresponded closely
with those found destroyed in cats without temperature control.

The disturbing factor lies in the fact that in the case of cats the tempera-
ture was always below normal, whereas in the cases reported here the
temperature was decidedly above normal. It seems clear, therefore, that in
man, at any rate, lesions in the same region may produce either hypothermia
or hyperthermia, and it also seems true that the area implicated in the
production of hyperthermia in man is also involved in the loss of temperature
regulation in cats.

R. M. S.

[66] Studies on pathological neuroglia in man (Studi sulla nevrogia pato-
logica nell'uomo).—G. CANZIANI. Riv. di pat. nerv. e ment., 1935,
46, 409.

In the studies of the histopathological alterations in the neuroglia demon-
strated in the brain in nine cases of senile dementia by Lugaro's bromide of
silver method it was shown that:
Hypertrophy occurs similar to that seen in progressive paralysis, but not so constantly. It may be confined to a few hypertrophied cells appearing amongst normal astrocytes which show no signs of hypertrophy.

Hyperplasia is also not so marked or constant as in general paralysis. There is no correlation between hypertrophy and hyperplasia or between the hyperplasia and the destruction of the cerebral tissue.

The splitting off of the dendrites is variable from case to case, and is perhaps connected with a process not directly related to the process of dementia praecox.

The author describes a degenerative process not previously recorded which he calls cystic degeneration. It consists in the formation of small discrete cavities confined to one special area. These tend to collect into areas like round bunches of grapes. They probably represent collections of lipoids which, with the methods of staining, appear as empty spaces.

The study of the splitting off of the dendrites shows that this process takes place in four stages.

R. G. G.

[67] The neuroglia in the basal ganglia in cases of general paralysis treated with malaria and in those not so treated (La nevroglia dei nuclei della base di paralitici progressivi malarizzati e non malarizzati).—V. Longo. Riv. di pat. nerv. e ment., 1936, 46, 508.

These observations show that whether the disease is in the first or second period, and whether treated by malaria or not, the appearance of the neuroglia does not differ appreciably, and that malarial therapy has very little influence on the neuroglial picture.

R. G. G.

[68] On some particular aspects of normal neuroglia which might be confused with pathological neuroglia (Su alcuni particolari aspetti della nevroglia normale confondibili con alterazioni patologiche).—G. Canziani. Riv. di pat. nerv. e ment., 1935, 44, 564.

The author has shown that splitting off of the dendrites may occur in the brains of animals which are fixed rapidly after death.

This artificial splitting involves a fragmentation of the processes associated with swelling and occurs in four phases: the first with the dendrites becoming fragmented, the second in which the dendrites show swelling up in the process of fragmentation, the third in which the cells appear amœboid since the swollen dendrites look like pseudopodia, the fourth in which the processes are destroyed and the protoplasmic bodies undergo a process of progressive corrosion.

R. G. G.
A study of the cerebral macroglia in status epilepticus (Studio sulla macroglia cerebrale nello stato di male epilettico).—F. Cardona and O. Meco. Riv. di pat. nerv. e ment., 1936, 47, 627.

The authors discuss two cases and conclude that the methods of Bielchowski-Cajal, Rizzo, Jedlowsky, etc., used for staining the cerebral macroglia of patients who have died from status epilepticus, do not show the regressive alterations of the glia which Gorriz and Perez, using the gold sublimate stain of Cajal, have described under the name of microclasmocytosis.

R. G. G.

On the pathogenesis of the diffuse glial reaction in cerebral tumour (Sulla patogenesi della reazione gliale diffusa da tumore cerebrale).—F. Cardona. Riv. di pat. nerv. e ment., 1935, 46, 748.

The author thinks that this glial reaction at a distance from the tumour is due to a reaction to a toxin produced by the tumour.

R. G. G.

A study on the post-mortem alterations in the macroglia of the dog studied by a new method of staining (Ricerche sulle alterazioni cadaveriche della macroglia del cane, studiate con un nuovo metodo di impregnazione).—M. Piolti. Riv. di pat. nerv. e ment., 1935, 44, 120.

Lugaro’s method was used on eight dogs. The experiments showed that the macroglia showed post-mortem changes as early as eight hours after death, but that they were yet highly resistant to these since the cells were still recognizable 168 hours after death.

R. G. G.

Remarks on experimental aseptic meningitis (Considerazioni sulle meningiti asettiche sperimentali).—C. Rizzo. Riv. di pat. nerv. e ment., 1936, 46, 373.

The author reviews the literature and describes his own experiments and concludes that almost any substance, even if it is isotonic, injected into the subdural space produces an aseptic meningitis with clinical symptoms and changes in the cerebrospinal fluid.

The humoral syndrome in the fluid may show immediate violent, but often ephemeral changes, according to the degree of toxicity and re-absorbability of the substance injected. After a day or two the fluid gradually
returns to normal and the cells are destroyed or altered. If the substance is injected into the arachnoid space the fluid becomes hemorrhagic to begin with. In addition to blood elements, cellular elements are found, especially eosinophil granulocytes. These reactions are important, since substances of various sorts introduced for purposes of diagnosis or therapy are likely to produce them.

R. G. G.

[73] On the possibility of cultivating the tubercle bacillus from the blood or cerebrospinal fluid of subjects of certain nervous system diseases (Sulla possibilita di coltivare il bacillo tubercolare del sangue e del liquor in alune malattie del sistema nervoso).—C. TRABATTONI. Riv. di pat. nerv. e ment., 1935, 44, 90.

The author applied Loewenstein's method to 16 cases of dementia praecox, two of disseminated sclerosis and two of chorea, but could get no confirmation of his results. Nor was any growth of tubercle bacilli obtained in five cases where they were present in great quantity in the sputum. He thinks, therefore, that the theory that the former diseases are tubercular in origin is to say the least of it premature.

R. G. G.

[74] Final considerations on experimental torula infection of the nervous system (Ulteriori considerazioni sulla torulosi sperimentale del sistema nervoso).—V. TRONCONI. Riv. di pat. nerv. e ment., 1936, 47, 504.

In a previous study of infection with Cryptococcus histolyticus it was noticed that there was a definite modification of the interstitial tissue both in the medullary grey and white matter illustrating some details not previously described.

The effects of torula infection in producing an ependymochoroiditis is described and the occurrence of fuchsinophil granular cells of Alzheimer is noted in relation to the splitting off of the dendrites from the neuroglia.

R. G. G.

[75] Paraphysial cysts.—A. J. MCLEAN. Arch. of Neurol. and Psychiat., 1936, 36, 485.

The paraphysis, which exists in many vertebrates, is present also as a vestigial remnant in embryonic and at times in postnatal life.
Its cystic enlargement forces it to sink through the membranous lining of the third ventricle as a pendulous tumour between the foramina of Monro. Its cytological picture is distinct; the stroma of the cyst-wall is composed of fibrocollagenous connective-tissue, and its lining of cylindrical and cuboidal epithelium. Its colloidal contents can be shown to give the pigmentary reaction of a lipochrome.

A fairly distinctive clinical syndrome is caused by the tumour, of which intermittent acute hydrocephalus is the most consistent element. The tumour has proved susceptible to successful total extirpation.

R. M. S.

[76] Calcium content of the blood serum during an epileptic convulsion.—M. Scott and A. W. Pigott. Arch. of Neurol. and Psychiat., 1936, 36, 590.

An estimation of the calcium content of the blood serum during a convulsion was made for 50 unselected, nonfasting institutionalized epileptic patients, and the value was compared with that of a control specimen taken between seizures. The type of epilepsy and associated factors, such as the race, the sex and the age of the patient, the length of time the disease had existed and the time of day and the presence or absence of a convulsion when the specimen was taken, had no appreciable effect on the calcium level, which was from 9 to 14 mg. per 100 c.cm. in 93 per cent. of the determinations, with an average for all specimens of 11.4 mg.

An adequate series of determinations made during a convulsion, with which the authors could compare their findings, was not available in the literature.

The determination of the calcium content of the blood serum during a seizure showed no appreciable difference from that made for the same patient between seizures. This study shows that hypocalcaemia is uncommon in patients with chronic epilepsy; a normal calcium content (from 9 to 11 mg. per 100 c.cm.) or hypercalcæmia (a content of from 11 to 14 mg.) was the usual finding.

R. M. S.


The writer's main results show that (1) the white cell count is very variable in epilepsy, its greatest variability being at the time of convulsion; (2) there is a definite leucocytosis; (3) this leucocytosis is associated with the occurrence of fits; (4) it is due in practically all cases to a relative increase in the leucocytes; (5) eosinophilia, traditionally associated with epileptic convulsions, occurred but rarely.
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The subsidiary findings were: (1) Eosinophilia is found in that minority of cases where the increased white count, associated with fits, is due to the polymorphonuclears. (2) When fits occur in groups there is a tendency for a low count to be associated with a period of fits. (3) The red cell-count and hemoglobin percentages vary, in association with the convulsions, but to a less degree than the leucocytes. (4) There is an increase in the erythrocyte count at the time of the fits. This may, or may not, be accompanied by an increase in the haemoglobin percentage. (5) There appears to be no definite relation between the type of cell count and the degree of frequency of the fits.

C. S. R.

[78] Relationship between the cerebrospinal fluid sugar and blood sugar in untreated neurosyphilis.—Purcell G. Schube. *Amer. Jour. of Psychiat.*, 1936, 93, 139.

A study of the simultaneous fasting cerebrospinal fluid sugar and blood sugar in untreated neurosyphilis is here presented. The cerebrospinal fluid sugar was within normal range in 88·2 per cent. of the cases and the blood sugar in 89·2 per cent. The ratio existing between them was within the normal range in only 51·2 per cent. of the cases of neurosyphilis, 51·5 per cent. of the cases of general paresis, 55 per cent. of the cases of cerebrospinal syphilis with psychosis, and in 41·7 per cent. of the cases of tabes without psychosis. This ratio was below normal in 18·2 per cent. of the cases of neurosyphilis, 12·7 per cent. of the cases of general paresis, 15·0 per cent. of the cases of cerebrospinal syphilis with psychosis, and 16·6 per cent. of the cases of tabes without psychosis. The ratio was above normal in 35·6 per cent. of the cases of neurosyphilis, 35·8 per cent. of the cases of general paresis, 30·0 per cent. of the cases of cerebrospinal syphilis with psychosis, and 41·7 per cent. of the cases of tabes without psychosis. Therefore, in 48·8 per cent. of the cases of untreated neurosyphilis there is definite evidence of some abnormality in the haemato-encephalic barrier to the passage of sugar.

C. S. R.


A familial case is presented which showed (1) diffuse demyelinization together with circumscribed lesions, resembling atypical patches of disseminate sclerosis; (2) within the diffuse demyelinization, persisting perivascular islands which were regarded as breakdown stages of the islands known in Pelizaeus-Merzbacher disease; (3) few remnants of 'concentric' structures.

The case seemed to invite discussion of the theory put forward by
Hallervorden and Spatz with regard to the spread of diffuse and disseminate sclerosis from the spinal fluid and bloodvessels respectively. This view is criticized in the light of comparative facts which are in favour of the vascular and particularly the venous system governing the spread of diffuse sclerosis.

C. S. R.


A case of familial amaurotic idiocy, unusual in many ways, is described. The child of a Gentile family, normal at birth, became very quiet at the age of six months, and showed no interest in his surroundings. He came under observation at the age of two years and four months; the patient died three months later. High degree of idiocy had been observed in his later life and persistent contracture of the right arm and leg; the left arm was fixed in an adducted position, athetoid movements were present in both arms and Babinski’s and Oppenheim’s signs in the right leg. No alteration was observed in the fundus oculi, the pupils reacted normally to light. At autopsy the brain volume and weight were very low.

On microscopical examination, the typical signs of amaurotic idiocy were observed, viz. accumulation of lipoid in all ganglion cells of the entire central nervous system. The dendrites of many ganglion cells were twisted in a cork-screw manner as observed hitherto only at an advanced age in a chronic disease. Throughout the brain, the grey matter presented the features of a high degree of so-called ‘status spongiosus.’ Nerve-fibres were completely demyelinated in patches in many parts of the central nervous system. On special investigation it was found that similar cases had occurred in the patient’s family.


Histological examination was performed in three cases of typical progressive muscular dystrophy and in one case of muscular dystrophy combined with a lesion of the pyramidal system. Typical changes were observed in the
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muscles as well as an intensive degeneration in the autonomic cells of the spinal cord and in the non-medullated fibres of the gangliated cord and also diminution of fibres and intensive degeneration in the non-medullated nerve-fibres of the muscles.

In some cases the sympathetic system was more damaged than the parasympathetic; in other cases the reverse was observed.

M.


In the group of disorders comprising Wilson’s disease and so-called pseudosclerosis the hereditary character of the degeneration is demonstrated exclusively by the changes in the liver. Brain lesions in such cases are a result of disordered metabolism arising from the primary disease of the liver. It follows that Wilson’s disease as well as family amaurotic idiocy does not result from a primary abiotrophy in the brain.

M.


Senile plaques were first described by Blocq and Marinesco (1892), who called them sclerotic plaques of neuroglia, a name which was later changed by Redlich to miliary sclerosis. The term ‘senile plaque’ was first applied to the lesion by Simchovicz.

In preparations impregnated with silver the plaque may be observed under medium magnification, as being composed of scattered clumps of irregular argyrophilic material; often in the centre there is a fairly homogeneous spheroid mass. In the immediate neighbourhood of this central mass the irregular clumps are usually less abundant than at the periphery. They tend to have a radial arrangement. In the outer zone of the plaque the arrangement of the clumps is very irregular.

The complexity of the histological appearance of the senile plaque is due to two main factors, viz., proliferation of living glial elements, along with disintegration of the necrotic plaques.

Morphologically some oligodendroglia cells in cases of senile dementia show a specific degeneration and develop into minute senile plaques. Here it should be remembered that the specific histochemical reaction gives conclusive proof that the degenerated oligodendroglia cells and the senile plaques are part of the same morbid process. The former represent an early
stage; the latter the final outcome. Specifically, mucicarmine stains selectively all senile plaques, and also those small elements corresponding to degenerated oligodendroglia cells.

Adequate studies of the histology of the mucoid degeneration are meagre at present, but it seems to be established that minimal quantities of mucin may be present or absent in a normal brain. In several morbid conditions, especially when the function of the oligodendroglia is impaired, degenerated oligodendroglia cells appear abundantly, possibly arising from the premyelin substance. Finally, they may become mucocytes, and a process of disintegration results in complete dysfunction. The writers consider that sufficient facts are at hand to warrant the following conclusion:

In the brain in cases of senile dementia oligodendroglia cells undergo a specific degeneration. They become mucocytes, or mucinization occurs, with an accompanying process of disintegration, and the resultant lesions are the senile plaques.

R. M. S.


To explain cases of congenital occlusion or stenosis of the aqueduct of Sylvius two main theories have been advanced: (1) An intrauterine infection causing a secondary proliferation of subependymal glia; (2) a developmental error, the fetal aqueduct becoming closed, much in the same way as the central canal of the cord closes, independently of any inflammatory process.

In the six cases reported by Roback and Gerstle neither of these explanations was applicable; there was no evidence of inflammation either in the subependymal zone or in the meninges, and the segregated glial proliferation described by the authors in their cases could not possibly be the result of an exaggerated narrowing of the iter.

Instead it is suggested that aqueductal atresia is a form of malformation, the sequence of events being somewhat as follows. During the course of normal development the cells that make up the brain tissue make their appearance as undifferentiated cells close to the ependyma. Adjacent to the ependymal layer is the mantle layer, from whose undifferentiated cells neuroblasts and spongioblasts are formed. These cells proliferate in the mantle layer and migrate towards the periphery. It is reasonable to conceive of the possibility that in the process of proliferation and migration some cells fail to migrate. These cells, instead of wandering toward the periphery, may in the course of proliferation be dislodged or even actively migrate towards the ependymal layer. The proliferation of cells in the ependymal layer interrupts and pushes the ependyma aside. Some ependymal cells may become entangled in the proliferating cellular mass. The glial proliferation may therefore be
interpreted as a developmental error caused by the abnormal proliferation and behaviour of embryonal cells.

R. M. S.

SENSORIMOTOR NEUROLOGY


Congenital stenosis of the aorta is not a very uncommon anomaly. Circulatory disturbances with brain involvement incident to this malformation may occur. Autopsy of the patient under discussion verified this fact.

The patient presented not only a congenital stenosis of the aorta, but also a bicuspid aortic valve. The latter is a common finding and a frequent site for the superimposition of an endocarditic infection. The frequent involvement of the aortic flaps of the mitral valves and the secondary bacterial invasion of such congenital anomaly of the heart are well illustrated in this case. It is to be noted that one of the terminal causes of death in this case was a subacute vegetative endocarditis.

The mental symptoms, the hemiplegic attacks and death of this patient occurred between the second and third decade of life. Clinically he presented cardiac signs of a congenital anomaly of the heart together with excessive pulsations in the vessels of the neck, the suprasternal notch and palpable pulsations in several intercostal spaces. There was a marked difference in the blood pressures of the lower and upper extremities together with definite signs of cerebral involvement. He conformed categorically to the 'text-book' description of cases of coarctation of the aorta. There were evidences of arteriosclerosis in spite of the patient's youth and a bilateral simple optic atrophy. There was no evidence of either congenital or acquired syphilis.

Aphasia, dementia, focal and bilateral paralysis resulting from previous attacks of hemiplegia, incontinence, athetosis and muscular contractions were some of the manifest neurological signs the patient presented. When one considers the various sensory and other motor disturbances which may have been present, but which could not be elicited owing to the mental state of the patient, it is at once apparent that almost any and all neurological complications may be encountered in a case of this kind.

The neurological signs and symptoms are not primarily neurogenic in origin, but are secondary to the vascular complications such as cerebral haemorrhage, infarct, embolism, aneurysmal rupture, encephalomalacia and cerebral arteriosclerosis. The greater part of the anatomical diagnosis in this case is concordant with such a contention.

Although congenital cardiovascular anomalies may or may not be associated with congenital degenerative diseases of the nervous or other systems, the characteristic findings of a stenotic aorta previously mentioned, even in the presence of a heterogeneous conglomeration of neurological signs