of the lenticular nucleus and of the anterior nucleus of the thalamus. The changes appeared to be an axonal reaction rather than a primary chromatolysis. There was no evidence of infection in the nervous system.

They found no changes in the cerebral or cerebellar cortex and no increase of glia cells throughout the cord.

In conclusion they state that the association of disease of the upper respiratory tract with the degenerative changes found in the nervous system in this condition, favour the view that it is probably due to an infection, the toxins of which affect the nervous tissues.

P. W.

SENSORIMOTOR NEUROLOGY.

[152] New ideas as to the genesis of transient hemiplegia and cerebral softening (Les idées nouvelles sur la génèse de l’hémiplégie transitoire et du ramollissement cérébral).—J. LHERMITTE. L’Encéphale, 1928, xxiii, 27.

An interesting exposition of the present status of the problem of evanescent cerebral attacks associated with old age and with arteriosclerosis. The author first discusses the vexed question of a possible nervous control over the cerebral arterioles, and quotes at length Florey’s work on the capillary reactions towards various types of stimulus. Passing to the clinical aspect of the subject, Lhermitte throws doubt upon the conception of vascular spasm in the production of transient hemiplegias; he inclines towards a hypothesis which has long been familiar to English medicine and which has lately received a fillip from the work of Naffziger, viz., the theory of local ischaemia from an attack of relative hypotension. Thus a state of transient hemiplegia is regarded as the result of two factors; first, the presence of diseased and partially occluded blood-vessels, and secondly, an incidental and temporary lowering of blood-pressure with a local relative retardation of the blood-stream. The importance of this view-point in determining the correct therapy is of course obvious, and stimulating measures (adrenalin, digitalis, saline injections) are advocated. Lhermitte extends this conception of relative hypotension so as to embrace the pathogenesis of encephalomalacia. He quotes the work of Foix and Ley, who demonstrated that—contrary to the previously accepted view—complete vascular occlusion is the exception in cases of cerebral softening.

These views will not prove novel to English neurologists. Although almost certainly the correct explanation of many instances, this hypothesis must not, however, be strained so as to include every case of transient ictus. It seems more probable that evanescent attacks are of varying origins, and that the morbid physiology differs according to the nature of the arterial changes, the presence or absence of hypertension, the age of the patient and other allied factors.

M. C.
Left hemiplegia and hemianaesthesia dolorosa in a case of CO poisoning (Hemiplejia y hemianestesia dolorosa izquierda en un caso de intoxicacion por el CO).—A. F. VAMAUER and A. BATTRO. Revista Oto-neuro-oftalmol., 1928, iii, 389.

As a result of poisoning with CO gas a patient developed a left flaccid hemiplegia, with a more or less complete thalamic syndrome, and also exhibited general symptoms of a toxic psychosis. The unilaterality of the neurological symptoms is unusual in cases of this kind, in which more commonly the corpora striata are involved, either by hemorrhages or by necrotic changes.


Some cases of cerebral haemorrhage in persons regarded as not having reached the degenerative period (ages 43, 50, 33, 40 respectively) are described with considerable detail; one in particular is subjected to the fullest pathological examination. The view adopted is that, in the absence of cardiac and renal disease and of hyperpiesis, the condition apparently must be ascribed to a certain thinness of the bloodvessel walls recognisable microscopically, and to a certain homogeneous degeneration therein, almost amounting to a necrosis, especially in the neighbourhood of the extravasations. No definite decision, however, on the question of whether these changes are primary or secondary, can be given. The haemorrhages were not a mere diapedesis. Mention is made of 'purpura cerebri.' Differential diagnosis is also discussed.


RAVID summarises briefly the various untoward reactions which have occurred during insulin hypoglycaemia, and describes a case of his own in which he observed as a reaction transient but complete hemiplegia on two occasions. The hemiplegia, ushered in on both occasions by left-sided numbness, progressed to a total, flaccid, left-sided paralysis with all the signs of a right capsular lesion. This reaction lasted on one occasion seven hours, on the other three hours, and disappeared leaving no residua. In this case, that of a man, age 34 years, atherosclerosis and cardiorenal disease could be excluded with reasonable assurance.

The author brings this phenomenon into line with other syndromes sine materie and mentions the transient hemiplegias occurring without organic change in tuberculosis, alcoholism, uræmia and pneumonia. It is interesting
to note that they are especially frequent in diabetes, and this fact emphasises the importance of demonstrating an actual hypoglycaemia in all so-called cases of insulin reaction. A definite hypoglycaemia was demonstrated on both occasions in this case.

The mechanism and nature of the action of insulin are discussed, and it is suggested that in all hypoglycaemic reaction there is a definite effect on the central nervous system. How this effect is initiated or what its nature is, is as yet matter for speculation.

P. W.


By echographia is meant involuntary copying of writing or print when the patient understands that he is to answer a written or printed question, not to copy it. Clearly several possible sources of error have to be eliminated before a case can be admitted into the category; above all, the investigator must make sure that the copying is not being done voluntarily.

Only a very few cases of this kind have hitherto been recorded. Sittig reports two personal cases which seem to be free from objections. The first was one of cerebral haemorrhage, with right hemiplegia and motor aphasia; the second, tumour of the left facial area. In the latter, echographia appeared as a transient symptom after operation.

An interesting and critical discussion of the symptom is given by the author, who advances reasons for regarding it as a cortical reflex, released by failure of transcortical inhibitions.

S. A. K. W.


The literature of decerebrate rigidity is reviewed and it is pointed out that this phenomenon depends on the integrity of a structure or structures between the midbrain and the medulla, since it does not appear until ablation has extended to a region below the thalamus; section of the medulla abolishes it, flaccidity of the limbs supervening. Experimental work on rabbits shows that the destruction of the inferior olives abolishes decerebrate rigidity. Three cases are quoted in which although the olives were intact their connexions with higher structures were interfered with.

R. G. G.


An exhaustive study of the literature is undertaken. Some 39 cases are studied and the author concludes that in spite of the vast amount of work done no very clear conception has yet been arrived at.
ABSTRACTS

The subject has been approached from every possible angle; perhaps the most recent psychogenetic approach is significant, though the author has noticed the conspicuous absence of the so-called typical epileptic personality make-up in certain cases. It is possible that a multiplicity of etiological factors has to be considered in genuine epilepsy, some being due to biochemical disorders while others may be ascribed to errors in the nervous system. With regard to the latter factor it may be we require more autopsy examinations of early cases before secondary changes have taken place. R. G. G.


A study of 100 patients indicated that, in the main, epileptics show a normal condition of acid-base equilibrium in the body fluids. Abnormality when present is in the direction of increased alkalinity. R. M. S.


The chief clinical features in the case recorded comprise: (1) transitory attacks of sensory aphasia, with jargonaphasia and jargonagraphia. These attacks gradually led up to a permanent state of defective speech, with loss of acquired (foreign) language.

(2) Uncinate seizures with dreamy states, peculiar hallucinations of taste and smell, tingling round the mouth, pain in the nose, and oesophageal spasm.

(3) Transient attacks of ataxia with dysmetria and intention tremor.

(4) A quadrantic homonymous hemianopia.

(5) Various transient visual phenomena:—

(a) Chromatopsia of a blue or violet tint.

(b) Bizarre distortions in the appearance of persons and objects.

(c) A particular feature which the author speaks of as "microteleopsia." The patient's surroundings seem to be transported far away, everything seems small and at a distance; the patient goes on talking but raises his voice and talks loudly in order to make himself heard.

Papilloedema was absent throughout the three years over which the symptoms had developed.

Although operation revealed a cystic tumour lying deep within the substance of the left temporal lobe, the exact location of the tumour is unfortunately not stated. M. C.

Attention is directed to the fact that varieties of tuberculous meningitis occur with much greater frequency than is usually supposed to be the case, and also that in not a few instances the condition is chronic and may, moreover, be recovered from. The diagnosis is made naturally by the discovery of the bacillus in the spinal fluid, but it is well to realise the usefulness of other present-day methods of examination. Reduction of sugar in the spinal fluid, with increase of protein and little change in chlorides, is highly characteristic, and the combination is by no means rare. Yet not a few cases of this kind improve with treatment. No real distinction is to be drawn between meningismus and meningitis; such differences as exist are quantitative, not qualitative. Clinically the occurrence of "toxic meningitis" is common enough. A description is given of personal cases, and others from the literature, in which tuberculous processes of different kinds have produced either an actual or a toxic meningitis, and in which amelioration or cure has taken place.

J. V.


A girl with extensive furunculosis of the body strained her back while performing artificial respiration. Four days later she developed a complete paraplegia of the lower extremities. Death occurred suddenly while she was being prepared for operation.

Pathological examination revealed an obliteration of the posterior epidural space at the level of the ninth to the twelfth thoracic segments by a granulation mass containing three small abscesses. The ganglionic portion of the spinal nerve roots, especially the posterior, showed perineuritis. The cord exhibited perivascular infiltration of the blood vessels of the anterior horns and scattered degeneration of the white columns, both types of change being confined to the lower thoracic region.

The author concludes that the case was an example of a haematogenous infection from an extensive furunculosis which had caused a severe non-inflammatory lesion of the spinal cord, with a clinical picture of so-called transverse myelitis.

To the reviewer the presence of inflammatory phenomena in the grey matter of the cord and nerve roots suggests a lymphogenous infection spreading to the dura from a staphylococcal focus which was described as being about one inch to the left of the spine in the lower thoracic region. This alternative is not discussed by the author.

R. M. S.
[163] **Endemic meningococcus meningitis.**—S. McLean and T. P. Caffey.

The authors report on a series of 136 endemic cases of meningococcal meningitis in infants. In their series 67-6 per cent. of the cases occurred in infants under one year of age. The age incidence is the same in both the endemic and the epidemic types. Seasonally the incidence is highest in April and September. They found that maternal nursing had apparently no protective influence and that the predisposing factors were over-crowding, poverty and poor hygiene in the home.

They stress the protean nature of the disease from the purely clinical side and point out that 50 per cent. of their cases were admitted later than one week from onset. Some of the best recognised signs of meningitis they found too variable to be of value in diagnosis. A tache or a positive Kernig or Brudzinski sign is of little value in infants under two years of age. The state of the tendon reflexes and the presence or absence of fever are also of little help.

The signs of greatest value were unexplained irritability, neck rigidity and strabismus. They also found of some value the presence of unexplained skin hemorrhages, a bulging fontanelle and the history of convulsion at the onset.

Above all they urge the necessity of immediate lumbar puncture in any suspicious case.

P. W.


Two personal cases are recorded in which hypersomnia and certain other symptoms were associated with basal lesions. In the first of these a carcinoma-like tumour was found in the infundibular region. The patient slept for days at a time, and often fell asleep while eating. After such periods of sleep came shorter periods in which he appeared unusually lively.

In the second case a cystic tumour about the size of a hazel-nut lay in the pes pedunculi, and reached as far forward as the infundibulum; it bulged into the third ventricle, pushing it over to the opposite side. For weeks the patient lay in an apathetic state, often fast asleep for days at a time, almost without waking. At other times he was less sleepy; he also often fell asleep at meals.

From these cases, and from other material in the literature, the author concludes that a centre for the regulation of sleep is situated in the vicinity of the third ventricle. He argues that this is a centre for the regulation of the alternating phases of sleep and waking, and that when it is disturbed these phases are disturbed. But the individual function of sleep, as such, is not disordered thereby.
Whether this argument is sound remains to be proved, in the opinion of the reviewer. The two cases here detailed were cases of severe destructive lesions; if nevertheless a hypersomnia was much the most prominent clinical symptom (as is specifically declared to be the case) then clearly sleep as such must be produced by mechanisms which are not situated in the regions that were destroyed. If sleeping and waking are "regulated" therefrom, the processes of each of these are attributable to mechanisms in activity in some other place than the vicinity of the third ventricle; but where this may be Dr. Pette gives no hint.

S. A. K. W.

[165] Arterial encephalography (Nouvelle technique de l'encephalographie artérielle; quelques cas de tumeurs cérébrales).—EGAS MONIZ. Presse méd., 1928, June 2, No. 44.

The author employs a solution of sodium iodide in 25 per cent. strength, and of this some 4 or 5 c.c. are injected in the following manner. The patient is prepared for operation and is placed in readiness on the radiological table. Under an anaesthetic an incision is made over the common carotid at its lower end and a ligature placed round the vessel. In the next place the external carotid is held ready for grasping with an ordinary artery forceps. The common carotid is immediately raised from its bed with the left hand, and the needle of the syringe containing the solution is inserted into its lumen with the right; as soon as ever the solution enters, the external carotid is cut off pro tem. with the forceps, the radiologist takes his photograph, the ligatures are removed, and the operation is finished.

The sodium iodide must be chemically pure and in sterilised solution, and should be at body heat when injected. Local anaesthesia with novocain suffices, but a general anaesthetic has also been used. The x-ray photograph must be of instantaneous exposure.

With this technique the author has obtained highly satisfactory pictures in no less than 25 cases, and in tumour cases, employing stereoscopic radiography, has been able to demonstrate displacement of arterial branches by the growth. He believes the method is of value and merits extensive trial.

J. V.

[166] The thermo- and glyco-regulating functions of the corpus striatum (Per la conoscenza delle funzioni termo- e glicoregolatrici del corpo striato)—S. D'ANTONA. Rivista di Neurol., 1928, i, 1.

Old questions of the central representation of body-heat functions have rather been lost sight of in the present-day concentration on the motor functions of the corpus striatum. In this paper is recorded a case of softening of the left putamen associated clinically with a rise of temperature on the opposite side.
of the body; the difference between the two axillae from the point of view of skin temperature averaged about one half or one degree centigrade during the better part of a fortnight, before death ensued. The case is taken to imply the existence of a heat-regulating centre in each corpus striatum, controlling the opposite half of the body. A discussion of the origin of the patient's glycosuria is also given.

J. V.


This is a long and not particularly lucid attempt to reconcile two different views of the pathogenesis of chorea, viz., the afferent theory of Bonhoeffer and the efferent theory of the Vogts and others. According to the former, chorea resulting from lesions of the superior cerebellar peduncle ('Bindearmchorea') gives a clue to the pathogenesis in a defect of afferent regulation; according to the latter, chorea results from lesions of the efferent striatal system, including the ansa lenticularis. The author, if the reviewer represents him correctly, has apparently found that a division of the main ansa lenticularis system runs underneath the lowest part of the optic thalamus in the vicinity of the nucleus ruber, just below the superior cerebellar peduncle; and he believes accordingly that the two views can thus be shown to be not contradictory, since a lesion in this area will involve both paths. What is missing in this long communication is any indication of the fundamental principles of the interpretation of pathological lesions of a destructive kind in relation to the clinical fact that choreic phenomenology is one of movement, not of paralysis.

S. A. K. W.


A case of streptococcal septicæmia with secondary involvement of the spinal cord, brain and meninges.

Among other symptoms the spastic smile, emotional instability, waxy rigidity of the arms, tremor of the head, arms and tongue and purposeless movements of the arms and hands, are thought to be evidence of localized involvement of the basal ganglia, which are not usually affected by pyogenic organisms.

R. G. G.
The syndrome of the superior cerebellar artery (Le syndrome de l’artère cérébelleuse supérieure).—G. GUILLAIN, I. BERTRAND, and N. PERON. Revue neurol., 1928, ii, 835.

This is a fine clinico-pathological illustration of a rare condition, considerably more so than lesions of the inferior cerebellar arteries. Clinically, the syndrome embraced three chief fractures, viz., involuntary movements on the right side, especially of the arm; a right-sided hemicerebellar symptom-complex; and on the left a dissociated hemianæsthesia of syringomyelic type. The involuntary movements are not given an actual descriptive name by the authors; they appear to have consisted of rather slow irregular contractions of flexors and of extensors, or of movements of circumduction of the fingers; they are said to have been less slow than those of athetosis.

The pathological state was one of softening of the superior aspect of the right cerebellar hemisphere and of the lateral aspect at the right side of the upper pons. The superior cerebellar peduncle and the lateral fillet were disintegrated; the contralateral red nucleus showed clearly the extent of this degeneration.

There was no involvement of either the pyramidal system or of any of the cranial nerves.


Two cases of rapidly ascending symmetrical flaccid paralysis with cranial nerve involvement form the subject of this paper. One of the cases was that of a previously healthy woman, age 48, the other that of a previously healthy man, age 27.

Both began with paræsthesiae in the toes which were followed by an ascending paralysis. In the first case there was generalised pain and tenderness at onset, followed by stiff neck, a positive Kernig’s sign and slight papillitis. There was also a bilateral peripheral facial weakness, right-sided keratitis and difficulty in swallowing. The cerebrospinal fluid showed 51 lymphocytes per c.mm. Deep tendon reflexes were abolished. The only sensory disturbance was tenderness on deep pressure and passive movement of the limbs. Recovery was complete in five months.

In the second case the onset and recovery were much slower. Marked atrophy coupled with R.D., loss of reflexes and fibrillation were present, but there were no signs of meningeal irritation. Postural and vibration sense was lost in the legs. Cranial nerve involvement was also prominent.

The author argues that a toxi-infective process beginning in the terminal fibres will inevitably spread by contiguity to some extent, and that a neuronitis
will be ultimately set up. He adduces the pain which is so common in poliomyelitis to support this contention, and suggests that peripheral neuronitis is the correct name for this type of case.

P. W.


This is a closely reasoned argument to the effect that the cause of the violent neuralgia and pains of many vascular states (angina, migraine, acrodynia, acrocyanosis, Volkmann's ischaemia, etc.) is not the relative ischaemia in itself, or the vasoconstriction, but the resultant venous stasis and relative engorgement. The argument is supported by many ingenious and cogent considerations, which merit attention. The author points out that venous blood is rich in carbon dioxide and products of metabolism, hence its irritative and toxic action; the effect may be exercised directly on the nerve filaments of the walls of the veins, or on the sympathetic plexuses and branches concerned.

J. V.


The author reviews in some detail the pathogenesis and symptomatology of the meralgic symptom-complex. The three cardinal signs of the syndrome he describes as being pain, paraesthesiae and some objective sensory loss occurring in the distribution of the external cutaneous nerve of the thigh. The picture may be complicated owing to abnormalities in the origin of the nerve, twigs being obtained from L 1 and L 3 in addition to the main supply from L 2, or owing to peripheral anastomoses with other cutaneous nerves.

He considers that the syndrome may result from lesions of an irritative or destructive nature in either of the first two neurones on the sensory path for pain—that is, either in the dorsal ganglia themselves or in the posterior horns. Furthermore he states that although meralgia is usually thought to be due solely to some peripheral abnormality it is often indistinguishable from some central lesions.

He goes on to discuss the various local and constitutional factors which may give rise to this picture. Trauma, directly affecting the nerve trunk in any part of its course, and various intra-abdominal conditions seem to be the commonest predisposing causes. He also quotes one interesting case which followed a zoster eruption in the distribution of the external cutaneous nerve, and mentions the fact that this syndrome may be the earliest symptom complained of in tabes.
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He gives two case histories in full, in one of which the meralgic symptom complex appeared after a bullet wound three inches below the left anterior superior iliac spine, in the other after long-continued pressure produced by leaning each day against a work-table.

P. W.


Rosenbaum records three cases of tabes dorsalis all occurring in children under twelve years of age. He stresses the fact that juvenile tabes dorsalis differs from the adult disease in several points. The most important of these are that years may pass before the deep tendon reflexes are lost and ataxia appears, that sensory loss and sphincter disturbances are seldom seen, and that mydriasis with loss of the light reflex is the commonest pupillary change.

Two of his cases showed gastric crises, rare in juvenile tabes. Herpes zoster occurred in one case. Ptosis was not seen, nor was there any type of mental disturbance.

P. W.

[174] **Compression of the spinal cord in Hodgkin's disease.**—G. A. Blakeslee. *Arch. of Neurol. and Psychiat.*, 1928, xx, 130.

The author reports a case of Hodgkin's disease of about thirteen years' duration with a complicating paraplegia developing after an interval of ten years. The signs of compression receded under Roentgen-ray therapy.

Reports of necropsies in the literature prove that the lymph-granula of Hodgkin’s disease can occur in the epidural space as a localised process, and it seems fair to assume Blakeslee's case had a similar localisation.

R. M. S.


In 1909 Heerfordt published three cases which showed a chronic enlargement of the parotid glands, a chronic iridocyclitis and a long continued low-grade fever. Two of these three patients also showed cerebrospinal paralyses. He proposed the name febris uveoparotidea subchronica for this symptom-complex. Numerous case reports, nearly all from Scandinavia and Germany, followed his paper. The authors record a typical case.

Their patient was a white boy, age 12 years, who had had mumps two years before this illness came on. The malady began with fever, which was rapidly followed by the development of bilateral iridocyclitis and enlargement of the parotids. There were no cerebrospinal paralyses. Improvement occurred
rapidly after radium applications, the technique of which is described. The course and symptomatology of the disease are variable. The most interesting complication is paralysis of cerebrospinal nerves, of which the facial is most commonly affected. Radiotherapy has given extremely good results as regards both the glandular enlargements and the iridocyclitis.

The authors discuss the numerous suggestions which have been put forward regarding the etiology of this condition. It is quite obvious that the disease has no etiological relation to mumps. Heerfordt himself considered a specific virus might be responsible. Since then tuberculosis and syphilis have been implicated by many authors, and yet others regard the syndrome as a sub-variety of Mikulicz's symptom-complex. One striking resemblance between the two is that familial types of both have been described.

That syphilis plays a part in producing either Mikulicz's or Heerfordt's syndrome has not yet been proved. It is certain that tuberculosis is the cause of some cases of Mikulicz's syndrome and it is probable that it plays a causal part in some cases of uveoparotitis, if a focal tuberculin reaction can be considered a reliable criterion. The tubercle bacillus, however, has never been demonstrated in the tissues of a case of uveoparotitis either histologically or by inoculation. The authors consider the condition to be a subvariety of Mikulicz's syndrome in which iridocyclitis is the outstanding feature.

P. W.


A case of otherwise typical amyotonia congenita is described in which the external ocular muscles are involved.

R. G. G.


The vibratory sensibility was tested in seventy-two persons between the ages of 10 and 90 years, who did not show any signs of organic disease of the nervous system.

It was found that the ability to perceive vibratory stimuli from the lower extremities becomes impaired in most persons after the fifth decade. The degree of impairment increases with age. This impairment is a result of diminished blood supply to the central and lateral portions of the columns of Goll in the thoracic region of the spinal cord, as a consequence of the generalised arteriosclerosis that occurs in the later decades of life.

R. M. S.
Voluntary extrusion of the eyeballs (Propulsión do los ojos voluntaria-
mente).—H. FERRER. Revista Oto-neuro-oftalmol., 1928, iii, 403.

This presumably genuine case is altogether remarkable and is probably unique. The subject is a young man who has the power of extruding either eyeball, or both, voluntarily to such an extent that it surpasses the degree of proptosis seen in the worst cases of exophthalmic goitre. The photographs accompanying the description of the case are nothing short of amazing.

According to the reporter of the case the "feat" is accomplished by contraction of the obliques and relaxation of the four recti.

S. A. K. W.

PROGNOSIS AND TREATMENT.

The preparation of serum from human donors recovered from polio-

The use of convalescent serum of both human beings and monkeys has been shown to be of value in the treatment of the disease whether naturally acquired or artificially produced. The quantity of blood obtainable from monkeys is insufficient for practical therapeutic purposes. Human donors are hard to obtain. The quantity of blood one can remove from an individual patient is strictly limited and only a small number of successive bleedings can be made from the same patient. The yield of serum is small—only about 150 c.c. being available as filtered serum from each bleeding.

The blood flowing from the convalescent's vein is collected in a vessel containing oxalate solution which is gently agitated during the whole venesection to insure that the anticoagulant is thoroughly mixed. The reds then are allowed to settle to the bottom and the supernatant plasma is syphoned off. A sufficient quantity of calcium chloride is added to the plasma and a clot quickly forms which, when complete, is separated from the side of the vessel with a sterile glass rod or platinum loop.

The specimens of serum from separate donors are arranged in groups and those of the serum of the same group are pooled, and filtered through Pasteur Chamberland F. candles into collecting bottles. Serum is classified as belonging to one of four groups according to the length of time that has elapsed since the donor suffered from the disease. Sterility tests are employed throughout, and safeguards are used to prevent transfer of infective disease from the donor to the recipient.

J. V.