Definite evidence of epileptic leucocytosis being influenced by digestion of food was lacking. Within the limits of physiological variation, the rise in the leucocyte count is the same in non-idiopathic as in idiopathic epilepsy. A remote effect is not produced by either grand or petit mal seizures on the white cell count in both idiopathic and non-idiopathic epilepsy. It seems that the leucocytosis frequently encountered in epilepsy is not due to the disease per se, but to some concomitant phenomenon. A connection between the leucocyte count in epilepsy and the presence or absence of an aura apparently does not exist. Observations failed to show any relation between onset and duration and the white cell count in epilepsy. The leucocyte count in epilepsy does not appear to be affected by the secondary anaemia which often accompanies this condition.

R. M. S.

SENSORIMOTOR NEUROLOGY.


This paper is an important contribution to our knowledge of the pathogenesis of transient cerebral attacks and also of cerebral softening in that it illustrates the trend of modern thought as applied to an important problem in cerebrovascular disease. The text of Spielmeyer's article is that pathologically demonstrable areas of ischaemia can exist in the brain unassociated with adequate anatomical changes in the blood-vessels of that part. It is suggested that there is a functional disturbance of the blood-supply due to a vasoconstriction of the arteries and capillaries lasting a longer or shorter time. The clinical expression takes the form of either transient or permanent focal manifestations. Spielmeyer points out that the ischemic zones may appear in stained preparations as a mere bleaching of the cortex; these may be overlooked on cursory examination and herein possibly lies the explanation of those cases of hemiplegia which are recorded as having no demonstrable pathological cause. It is interesting to note how the clinician—who has been long misled by the negative findings of the physiologist—is gradually returning to the conception of a vasoconstrictor process in the cerebral blood-vessels as a pathogenic agency.

M. C.


A useful review of the whole question of angiospasm in regard to the nervous system leads to the following generalisations.
1. The etiology of angiospasm is diverse; it includes local arteritis (not altogether definite), toxic agencies, endogenous or exogenous; and constitutional spasmophilia (Raynaud’s disease, migraine).

2. The pathogenic role played by the vasomotor system is doubtful, largely because of the insignificance of vasomotor innervation of cerebral vessels, especially in their terminal branches. Exogenous and endogenous poisons alike probably act directly on smooth muscle fibres. Some paroxysmal angiospasms are perhaps the expression of anaphylaxis.

J. S. P.

[94] The clinico-anatomical syndrome of lesions of the anterior choroid artery
(La sindrome anatomo-clinica conseguente a lesione dell’arteria coroidea anteriore).—U. Poppi. Rivista di Neurol., 1928, i, 466.

Clinically, a lesion of the anterior choroid artery reveals itself by (1) hemiplegia, (2) hemianesthesia, (3) lateral homonymous hemianopia, (4) vasomotor change (including edema), (5) the ‘thalamic’ hand. The artery supplies the posterior limb of the internal capsule, the thalamus (in part), the globus pallidus (in part), and also the external geniculate body and part of the anterior commissure.

S. A. K. W.


A careful histological examination of the wall of each of five aneurysms found in three cases, has been carried out; this has included the examination of serial sections. In one, the smallest aneurysm, a definite localized defect was found in the muscular and elastic coats. As no syphilitic, mycotic, or traumatic origin could be found, the hypothesis of Eppinger that these aneurysms are congenital was apparently upheld and confirmed by histological examination. Additional confirmation of this was present in one case, when other vascular maldevelopments were found, such as co-arctation of the aortic isthmus, a bicuspid aortic orifice, and fenestration of the mitral cusps.

E. A. C.


A case of the above is described, with post-mortem confirmation. The author seemingly thinks it the first case in the literature showing Sherrington’s phenomena of decerebration and the tonic neck and limb reflexes of Magnus and da Kleijn observed during apoplectic coma. It demonstrates the possibility of diagnosing from the above symptoms the participation of the upper
part of the brainstem in the lesion, as well as of the occurrence of the reflexes of Magnus and de Kleijn without the retention of the wholly or partially intact sensorium. A certain degree of rigidity is necessary before these reflexes can be demonstrated.

Pyramidal lesions are not, as some think, essential for the production of these symptoms, but lesions of the basal ganglia alone without involvement of the pyramidal tracts cannot cause them. Ordinarily such lesions do not produce tonic reflexes in apoplectic coma and this only occurs if the brain stem is also involved. Experimental work places the lesion in the region of the red nucleus. The case shows that when the tendon reflexes are maintained in deep coma the arc does not go through the cortex; their preservation probably depends on the same medullary centres as are responsible for decerebrate rigidity and these have much the same localisation in man as in animals. Finally, early contracture at the time of a stroke probably depends on the same lesions in the brainstem.

R. G. G.


In these two valuable contributions the authors have reported in anatomical and clinical detail eight examples of an akinetic syndrome associated with senility. The cases are considered in three groupings: the first subdivision includes cases illustrating a hypertonic-hypokinetic symptom-complex of the arteriosclerotic-rigidity type. The putamen was the seat of severest pathological change although lesions were demonstrable throughout the corpus striatum and also in the capsule of the olives and in the nucleus dentatus. To the second group are relegated those cases which were characterized clinically by a combination of rigidity and hypotonia. Pathologically the cases proved to be of the nature of a combined strio-pallido-dentato-rubro-peduncular system disease, in which the nucleus ruber was 'isolated' (in the sense of Munk) from both afferent and efferent influences. Group 3 contained two cases in which cortical or subcortical lesions co-existed and were notable clinically on account of the added manifestations of apraxia, agnosia, echolalia, and by marked psychic changes. The second of the two patients belonged clinically to the cadre of Jakob's 'senile Vorsteifung.'

In their second paper the authors review in interesting fashion the pathophysiology of the motility disorders of advanced age. A valuable discussion
centres round their sixth case, in which a state of chronic deforming arthritis was associated with a Parkinsonian syndrome. The correlation of these two morbid states is fully discussed and the authors conclude that joint deformities of cerebral (extrapyramidal) origin do occur; in outward appearance they exactly resemble the changes of peripheral causation met with in chronic progressive polyarthritis.

M. C.


The author has studied in a series of cases of hemiplegia the blinking reflex excited by the trigeminal (contact with the cornea or conjunctiva) and the blinking reflex of optical stimulation (strong light stimulus or threatening visual impressions).

Cases with hemianopia or corneal insensitivity were excluded from this study. The results in the other patients were complex; in some cases both types of blinking reflex were diminished in the homolateral eye. In other instances, only one of the reflexes was reduced, while in a few cases the blinking reflex for light was exaggerated. It is tentatively suggested that the optical blinking reflex may have a duplex character with distinct anatomical bases; thus the blinking reflex for light stimuli is probably elicited via the brainstem only, while the blinking response to threatening danger entails the implication of the visual cortex.

A practical point arises from this study: the usual practice of testing for hemianopia in a semiconscious or stuporose patient by means of threatening gestures towards the eyes from various angles is thus shown to be an untrustworthy measure. Loss of the usual blinking response does not necessarily mean hemianopia, and, conversely, lively optical blinking responses have been noted in cases with hemianopia.

M. C.


Unusual complications of epidemic encephalitis in respect of the voice and of speech functions generally have frequently been noted, but those here reported are rare if not unique. In the first case the patient exhibited an irresistible impulse to sing, and, in less degree, to read aloud; in the second case, the impulse was to speak in a much louder voice than usual (this was not, however, an encephalitic but an arteriosclerotic case).
The suggestion is made that the hyperodia signifies the release by disease of a "primitive psychomotor automatism," though what is meant by this term is not explained; the megaphonia is attributed to a release of function of both a pyramidal and an extrapyramidal component in voice production.

S. A. K. W.

The distribution of motor apraxia in the segments of the body
(Zur Frage der Verteilung der motorischen Apraxie auf die Körperteile).

It was long ago observed by Hughlings Jackson that certain motor phenomena were prone to develop in one part of the body rather than another. The usual sites for the commencement of Jacksonian epilepsy are forefinger and thumb, face, and foot (great toe). Similarly, in hemiplegia those muscular groups are most severely affected whose function is 'most voluntary,' and those least affected whose function is 'most automatic.' Again, in cases of 'phantom limb' after amputation those parts mostly take a share in the phantom residuum which have most small muscles (hand, foot), while more centrally situated groups participate little or not at all.

The authors apply these generalisations to the question of motor apraxia and conclude from their observations that the same rules apply in respect of that syndrome; isolated apraxia develops in the upper extremities, or in the face; if it implicates the whole body musculature then it is more pronounced in specific groups, as above, than in others. Clinical cases are cited in support of their conclusions.

S. A. K. W.


The authors refer to a paper by Hugo Meyer in which attention was called to the prevalence of a peculiar form of meningitis in children, the exciting cause of which was the Koch-Weeks bacillus. They themselves record three cases, with two autopsies.

The patient in the first case was a little girl of twenty months, who had been febrile for four or five days before symptoms of meningitis were apparent; then followed rigidity of the neck and Kernig's sign, somnolence and unconsciousness, and finally tonic and clonic spasms. Death occurred on the seventeenth day of the illness. Cerebrospinal fluid findings:—fluid under increased pressure; turbid; dense fibrin clot after long standing; cells, 1800 per c. mm. (polymorphs); Nonne and Pandy reactions strongly positive.
The second case was that of a male infant, seven months old, whose illness began with high fever and convulsions; three days later, he was admitted to hospital with the clinical appearances of 'Broncho-tetanie'; the first meningitic symptoms were seen on the sixth day of the illness and death occurred three days later.

The fluid findings were exactly similar to those in the previous case.

The third patient, a little girl of two years, began her illness with vomiting and a rise of temperature. A few days later neck rigidity and Kernig's sign were present and the spinal fluid was turbid. A month after the beginning of the illness convulsions began and consciousness was lost; death did not occur till the end of the seventh week. The temperature was never high, ranging between 38° and 39° C.

In all three cases bacteriological examination of the cerebrospinal fluid by smear and culture revealed organisms having the characters of the Koch-Weeks bacillus.

Autopsies were held in the first two cases; pus was abundant and poured out when the skull was opened. Patches of pressure atrophy were present in the dura. The leptomeninges were inflamed, with occasional circular hæmorrhages; over the anterior and lateral parts of the base of the brain and over the posterior parts of the convexity the leptomeninges were covered by a thick layer of viscous clinging pus of a striking yellow-green colour; it lay over the surfaces of the gyri as well as in the sulci, thus reproducing the brain-contours; the gyri were flattened. The ventricles were dilated and filled with turbid fluid. In the second case the mucous membrane of the nose and ethmoid sinus was inflamed and swollen, had hæmorrhages within it, and was covered with purulent yellow mucus. In Case 1 the left middle ear also contained yellowish pus. Smears taken at the autopsies revealed the same organism as had been found in the spinal fluid.

It may be that the preliminary clinical stage before the appearance of symptoms of meningitis corresponds to the development of the infection in the nasal passages.

J. P. M.

[102] Amaurotic family idiocy and general lipoid degeneration.—B. Sachs.
Arch. of Neurol. and Psychiat., 1929, xxi, 247.

Sachs reviews the pathology of amaurotic family idiocy in the light of recent research. His paper contains many points of interest which deserve careful study.

A few years ago Bielschowsky insisted that the pathogenesis of this disease must be linked with the source of lipoid substance within the nerve cells. Acceptance of this view implied opposition to the conclusions of Schaffer, who supposed that the lecithinoid granules found with startling uniformity in all the nerve cells of the central nervous system were to be regarded as products of degeneration. In the meantime, clinical and pathological studies
have revealed a surprising relationship with another group of diseases—with Gaucher's disease and with a familial and fatal disease of infancy showing great predilection for the Hebrew race and bearing some, if not complete, resemblance to Gaucher's disease. This infantile form is a lipoid cellular splenomegaly or lipid histiocytosis, and is now known as the Niemann-Pick type, in contradistinction to the Gaucher type. Both types would appear to be variants of a general lipoid cellular degeneration with enlargement of the spleen and liver. The Gaucher type may occur at any age. The Niemann-Pick type terminates fatally before the end of the second year. In the Gaucher type, the lipoid invasion affects the reticular cells and the connective tissue cells, the so-called histiocytes. In the Niemann-Pick type, the same invasion occurs not only in the entire reticulo-endothelial apparatus, but also in the elements of all other body tissues—in the muscles, in the glia, in the ganglion cells and in all epithelial cells, even in the cells of cartilage. Sachs suggests that if the similarity of these two types is to be acknowledged, the Gaucher type might be called the juvenile, and the Niemann-Pick type the infantile form of this splenomegaly, thus emphasising the resemblance of these forms to the two types of amaurotic family idiocy. The underlying disease process in all these forms is evidently a profound and primary disturbance of metabolism and it is Pick's contention that in Tay-Sachs disease the same disturbance in lipoid metabolism is the predominant factor. When this metabolic disturbance affects the entire body, the Niemann-Pick type, a universal disturbance of lipoid metabolism, is developed; the Tay-Sachs type is a disturbance of the same kind restricted to the nervous system.

In his last publications Pick recorded several cases of the Niemann-Pick type in which he made a complete examination of all the organs, including the central nervous system; he also recorded that in one of these cases, easily recognised as a form of the Niemann-Pick type, all of the cardinal symptoms of the Tay-Sachs type were present. Before deciding whether or not one is to accept the relationships of amaurotic family idiocy to the Niemann-Pick type, it will have to be shown that the symptoms of amaurotic family idiocy occur in a relatively large number of cases of the Niemann-Pick type, and it will be still more important to examine all the organs of a child dying of amaurotic family idiocy to prove that in those cases the histological cellular changes are similar, as Pick believes, to the cellular changes that are now so well known as occurring in every part of the central nervous system.

R. M. S.


The time-honoured controversy as to the influence of trauma on syphilis of the nervous system is here again discussed and illustrated by a number of cases.
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In the author's experience exacerbations of existing neurosyphilitic symptoms following trauma—even if the latter has been relatively severe—do not persist. The chief result of such traumata is the development of general nervous symptoms. In four cases in which local symptoms arose, two responded to treatment; in the third case the trauma followed neurosyphilis, and in the fourth the symptoms of the two processes were independent of each other.

Latent syphilis can light up spontaneously at any time, as well as in consequence of trauma. The usual effect of trauma is to produce a temporary exacerbation of meningeal processes. Cases with obvious existing syphilitic symptoms and with only latent syphilis react in the same way where trauma is concerned; hence it is well not to overestimate the effect of the latter in the production of syphilitic symptoms.

J. S. P.


A clinical presentation of an unusual set of symptoms occurring in a young man. The outstanding features were:

1. Bitemporal pallor of the optic discs with conservation of vision.
2. Pupillary inequality; ptosis; paralysis of the pterygoids and masseters; bilateral facial palsy; nerve deafness; paralysis and loss of sensation of the palate and pharynx. Atrophy and fibrillation of the tongue.
3. Generalized wasting, especially the peroneal and scapular groups, with fibrillation. Reaction of degeneration.
4. Exaggeration of the tendon jerks with right-sided ankle clonus and Babinski response.

There was no abnormality in the spinal fluid and the Wassermann reaction was negative.

M. C.


In 1859 Landry described a clinical picture, characterised by flaccid paralysis of all limbs coming on quickly and with distinctive sequence, the paralysis affecting first the lower extremities, then the upper, then the muscles innervated by the cranial nerves and finally the diaphragm; disturbances of sensation are absent or at any rate slight. In this paper two cases approximating to the Landry type are recorded with post-mortem findings. The first was that of a man of 26; the symptoms began with numbness in the toes of the right
foot and paralysis gradually spread upwards, the legs being completely paralysed by the sixth day; by the ninth day the facial muscles were weak and by the eleventh swallowing had become difficult; arm movements were at that time still possible. All tendon jerks were absent. There was no disturbance in micturition and defecation. Sensation was somewhat impaired generally over the lower limbs, and on the outer sides of the legs there was definite loss of appreciation of touch, painful stimuli and temperature. On the fifteenth day of illness paralysis of the diaphragm was apparent, and on the following day the patient died of bronchopneumonia. Post-mortem the findings were those of an acute infection: the spleen showed acute enlargement; histologically there were numerous inflammatory foci round the vasa nervorum, and the interstitial tissues of the nerves were swollen and oedematous: the myelin sheaths showed granular degenerative changes. The changes within the nerves were particularly noticeable in the proximal parts, but in the spinal cord no distinctive changes were found. In the nerve sections numerous Gram-positive encapsulated diplococci were discovered in the foci: they were apparently pneumococci and the authors of this paper regard them as the cause of the polynévritis.

In the second case, the patient, a young doctor of 25, died on the thirteenth day. His symptoms had begun with pains in the legs and back and with fever. The Wassermann reaction was strongly positive. On the fifth day the legs began to feel numb and by the ninth day the legs were completely paralysed; the arms were weak and the face was paralysed on the right side. The next day all limbs were paralysed and passive movement of them caused pain; the reflexes were all absent. No satisfactory examination of sensation could be made. On the twelfth day the pharynx and diaphragm became paralysed. Post-mortem there were found septic bronchitis and tonsillitis. Histological examination demonstrated perivasal inflammatory foci in the nerves and in the brain and other organs, the process being one of extensive distribution. As in the previous case, Gram-positive diplococci were found in great numbers.

While the clinical course of these cases in many ways resembled that of Landry's paralysis, we think they would be better described in view of the histological findings as acute polynévritis. Though the limits of the category of 'Landry's paralysis' are still not definitely laid down, the tendency is to restrict the term to cases without sensory involvement and showing after death changes in the anterior horn cells, but little or no inflammatory reaction. At the same time, the cases here recorded are of interest and the presence of organisms within the inflammatory foci is important.

J. P. M.


A female, age 26, had a period of diplopia and weakness of the arms lasting several months, ten years before coming under observation. Two years before
admission her legs became stiff and weak. On examination a spastic paraparesis was found with nystagmus and intention-tremor of the extremities; the tongue deviated on protrusion. The changes in the spinal fluid indicated compression. Later she complained of headaches; papilloedema developed, and cutaneous sensibility became lost below D6. At autopsy the following changes were noted:

1. A large endothelioma behind the medulla and upper cervical cord.
2. Chronic arachnitis fibrosa cystica in the region of the seventh and eighth thoracic segments.
3. A gliomatosis with central necrosis in the sixth and seventh cervical segments.
4. Numerous osseous lamellae imbedded in the arachnoid.

M. C.


This comparatively uncommon disease has aroused interest recently and the author records eight such cases. He reviews the symptomatology and points out the late onset of sphincter disturbance; he lays stress on the value of manometric readings at lumbar puncture for the early diagnosis.

E. A. C.


Intramedullary cysts may occur either above or below a tumour of the spinal cord as a result of cystic degeneration of the tumour, softening of an associated central gliosis, dilatation of the central canal, or foci of necrosis from vascular changes at a distance from the tumour.

In the author's case the cyst involved the entire cervical cord and was found above and separate from an intramedullary sarcoma. It was under marked tension and large enough to produce a spinal block. The tumour was diagnosed as a sarcoma but may possibly have been a hæmangioma or a hæmangioblastoma.

With regard to the mechanism of the cyst formation, it was not found possible to draw any definite conclusions. There was no indication that it was caused by cystic degeneration of a neoplasm, and a cyst of this size and under such tension could hardly be of the type associated with a gliosis. Whether due to a dilated central canal or to necrosis from distant circulatory changes, it appeared probable that the cord tumour caused a stasis of cerebrospinal fluid and contributed to the formation of the cyst and the tension of the fluid within it.
THREE cases are fully described and the literature is reviewed. The author concludes that in syringomyelia the importance of trauma is subordinate to the predisposition in the tissues, which may be either congenital or acquired. Vertebral deviation is important since this may be for a long time the only symptom of a developing syringomyelia. It may be due to an osteoarthritic atrophy or to muscular weakness causing distortion.

The total anaesthesia frequent in syringomyelia may be due either to a lesion of the posterior columns of the cord or to one in the grey matter which may occur at a considerably higher level. There may be multiple lesions irregularly distributed; the frequency of their unilateral character points to a congenital origin.

Deep radiography is the only therapeutic measure available to-day and acts by its power of fixing and resolving gliomatous processes.

R. G. G.

FOUR to five days after an inoculation with antityphoid serum, a man of 21 developed a quadriplegia which became complete in the course of 24 hours. The patient died of respiratory failure 60 hours later. Autopsy revealed complete necrosis of the spinal cord mainly in the region of the cervical enlargement. There were marked vascular anomalies in the nature of haemorrhages and thrombosis, but no signs of neuroglial reaction. The authors review the literature dealing with the neurological complications of antityphoid inoculation.

M. C.

THE two cases herein described are of considerable interest, seeing that they belong in reality to the cervical variety of disseminated sclerosis, and present the usual symptom-complex of that particular group. In addition, however, typical tetany-like seizures occurred (in the first case) mainly in the right arm and hand, but at first also in the left. Trousseau's phenomenon was positive. An elastic bandage round the right arm brought out tetanoid spasms in a few
seconds, as did pressure on the ulnar nerve. Yet other usual manifestations of tetany were wanting. The calcium content of the blood was found on two occasions to be within normal limits. More or less identical clinical observations were made in the second case.

The author examines in turn various theories to account for the peculiarities of his two cases. He has not found a similar combination of unilateral tetany with cervical cord symptoms in other recorded instances of so-called cervical disseminated sclerosis. He notes in particular the absence of any assignable cause for the tetany and of any of the usual accompaniments. After a consideration of several analogous cases of tetany accompanied by symptoms of involvement of the central nervous system, he concludes (as the reviewer thinks, on inadequate grounds) that "the site of origin of the hemitetany in our two cases lies in the subcortical ganglia"; and further that "the disorders both of deep sensibility and of tactile gnosis are referable to lesions in the subcortical ganglia and the internal capsule."

S. A. K. W.


The term sciatica, as it is generally employed, is misleading. It tends to direct attention away from the most common and remediable underlying cause of the various clinical conditions characterised by pains in the lower part of the back and limbs. The use of this term should therefore be discontinued, and an effort be made to determine the underlying cause and to designate the condition accordingly, and as being associated with the sciatic syndrome.

In the absence of a polyneuritis or polyradiculitis from whatever cause (toxic, infective, metabolic, constitutional, vascular, etc.), a primary mononeuritis or radiculitis of the sciatic nerve is extremely rare. In practically all the authors' cases the sciatic syndrome was secondary to some pathological process in osseo-arthritic structures or their contiguous soft parts in the region under discussion. Cases in which the patients have paroxysmal attacks of lancinating or shooting pains in the lower part of the back and in the lumbosacral distribution without objective evidences of involvement of the lumbosacral roots or nerves, or of the osseo-arthritic structures or their adjacent soft parts (muscles, tendons, fascia, etc.) in this region should be designated as "sciatic neuralgia." The presence of only three such cases in a series of 317 indicates the rarity of this condition, notwithstanding that most authors emphasize its frequency and employ the term 'sciatica' as if it were synonymous with 'sciatic neuralgia.'

By far the largest number of patients presenting the sciatic syndrome show demonstrable evidence of involvement of the osseo-arthritic system or of the
contiguous soft parts in this region. This group may be divided into two sub-
groups: (a) Cases without demonstrable signs of organic nervous disease. In
the absence of such signs, these cases present an orthopædic problem, and for
convenience they may be designated 'orthopædic cases.' (b) Cases with
demonstrable evidence of involvement of the lumbosacral roots or nerves. De-
pending on the nature and distribution of the involvement of the nerve they
may be designated as cases of lumbar, lumbosacral or sacral radiculitis. This group included 64 per cent. of the authors' material.

Except in such rare cases as sacralisation of the fifth lumbar vertebra, calcification or ossification of the soft parts, or in primary or metastatic neoplastic or tubercular processes in the spine or pelvis, positive X-ray ob-
servations of spondylitis or arthritis are not conclusive as to the etiological relationship of these observations to the sciatic nerve.

R. M. S.

and Psychiat., 1929, xxi, 392.

In papers devoted to sciatica attention is usually concentrated on the sensory
phenomena, and it is often assumed that neuralgia of the sciatic nerve leaves
the motor functions entirely or almost entirely undisturbed. In reality, this
is not true, for in this disease there is a whole series of motor phenomena
which are of great diagnostic importance. Of these the Kernig sign is the best
known. The Lasègue contralateral sign, often confused with the first-named,
consists of the following: the raising of the unaffected stretched leg brings on
pain on the affected side, mainly in the ischiatic area, sometimes in the lumbar
region, and sometimes down the entire leg. Flexing the head (the Lindner
sign) occurs as follows: if enforced passive flexion of the head to the chest is
effecte in a recumbent or sitting posture with outstretched legs, pain arises in
the lumbar region, in the ischiatic area, and sometimes down the entire leg
on the affected side. In acute cases this symptom is clearly manifested in a
recumbent posture; in subacute cases it may fail to appear, but it will do so
if the patient is made to sit up in bed with the legs outstretched. Closely
allied to the Lasègue sign is Neri's sign of bowing. When the patient, in a
standing posture, is made to bow, he flexes the knee on the affected side as if
curtseying; simultaneously a sensation of pain appears in the leg. The
author concludes that these phenomena are of a reflex nature and are immediately
connected with the pains arising from tension and irritation of the nerve and
roots. Besides this, the immediate irritation of the motor parts of the roots
apparently plays a part in their origin.

R. M. S.

[114] Unilateral neurofibromatosis of the cranial and deep cervical nerves.—

In the case reported there was an enlargement of the left side of the face due
to a myxomatous condition of the soft parts; the bony framework was actually smaller on the affected side than on the normal. In the left upper eyelid, the left cheek and the left side of the neck were numerous discrete, circumscribed, freely movable, painless nodules. These tumours were composed of nerve fibres and connective-tissue cells. The local interstitial proliferation of fibroblasts constituted the major portion of the tumour and dissociated the nerve fibres.

R. M. S.


Twelve further cases of this disease are added to the literature. All the cases completely recovered so no histological investigation was possible. The suggestion made by Greenfield and Paterson that the disease is a polyneuritis has been upheld, as in all cases there was a diminution in muscular power and sensation peripherally: no case, however, showed the inability to hold the head erect as described by the British authors. No common etiological factor was found; but the author considers that the condition may be infective (following infections of the respiratory apparatus) and not of the nature of deficiency disease.

E. A. C.


Though intermittent claudication may be a symptom of any form of arterial disease, usually it is due either to arteritis obliterans or to arteriosclerosis of all or a portion of a peripheral vessel. Davis reports ten cases in which the condition was a symptom of arteriosclerosis. Only two of the ten were women—a fact which emphasises the well-known greater occurrence of the condition in men. Transitory neurological signs in the precise form described by Dejerine were not found, but it was possible to distinguish two outstanding and contrasting spinal syndromes: (1) a predominantly spastic paraplegic syndrome, and (2) a predominantly poliomyelitic syndrome. In each there was a moderate involvement of the posterior columns, as evidenced by a disturbance of vibratory sensation. Impairment of bladder control was present and was conspicuously absent in another with a poliomyelitic syndrome of severe type.

R.M.S.


The rare disease known as periarteritis nodosa is commonly considered to be clinically recognisable by the following triad of symptoms: (1) a chlorotic
marasmus; (2) polyneuritis and polymyositis of varying intensity; (3) symptoms referable to the intestinal tract (abdominal pains, diarrhoea, vomiting, melena, perforation and peritonitis). In addition, frequent slowing of pulse, albuminuria, and fever deserve mention.

An interesting case of this kind is described in full; the condition of obscure polyneuritis was eventually diagnosed during life as being due to periarteritis and the surmise corroborated by pathological examination. In the central nervous system the chief changes consisted in the involvement of the smaller vessels and precapillaries of spinal cord and of cerebral cortex, as also of the capillaries, in alterations of ganglion cells and in diffuse glia proliferation in various places throughout the cortex. As for the peripheral nerves, acute and chronic changes were visible in the vessels of epineurium and perineurium; no parallelism was discovered between degree of vessel change and of local neural degeneration. In liver, spleen, kidney, pancreas and other abdominal viscera the typical picture of periarteritis nodosa was revealed.

S. A. K. W.


As well as recording eight cases of angina pectoris treated by paravertebral injection of alcohol a review on recent work dealing with cardiac pain is included. Operative interference has on the whole proved of little value. Jonnesco failed to bring relief by extirpation of the entire cervical chain along with the first thoracic ganglion; Danielopolu sectioned the cervical sympathetic chain above the stellate ganglion together with the vertebral nerve as well as a nerve which joins the superior cervical ganglion to the cranial nerves; he also removed the cervical sympathetic chain, cut the vertebral nerve, all the branches leaving the vagus to enter the thorax, and the rami communicantes which joined the inferior cervical ganglion and the first thoracic ganglion to the last pair of cervical nerves. None of these operative procedures was entirely successful. Experimentally Jonnesco showed that the cervical sympathetic chain does not convey pain discharges from the heart and aorta. With these facts before him the author examined each of his cases from the neurological standpoint and found that the area of skin supplied by the spinal segments C8 to D6 or D7 was hypersensitive. Accordingly, he suggested that pain impulses from the heart and aorta passed via the posterior roots of the above spinal nerves to the spinothalamic tracts. He then proceeded to inject these roots and ganglia with alcohol and in all cases produced relief from pain for several weeks up to several months. This method appears to be the most hopeful for the cessation of pain in angina pectoris.

E. A. C.

This reflex was first described by Moro in 1918. He found that on placing an infant on a table and then forcibly striking the table on either side of the child, a motor reaction was obtained. The arms were suddenly thrown out in an embrace attitude, describing an arc, and tending to approach one another with a slight tremor; the fingers were at first spread and then closed. Similar movements were observed in the lower extremities. A study of this reflex was made in 300 normal infants and in 21 sick children. It was found to be strongest during the first two months of life, to diminish in intensity from then on and to disappear at the end of the fourth month. Absence on one side suggests either a paralysis or injury, while persistence of the reflex above the age of six months signifies cerebral injury.

E. A. C.


One hundred and fifty-three normal and 121 patients were submitted to examination with the differential pupilloscope of Hess. Of the 121 patients only 31 gave no evidence of syphilitic infection. Among the syphilitic was a rather large number, diagnosed as merely neurotic or neurasthenic, in whom the pupilloscope proved syphilis to be manifest, and in each instance serum or fluid Wassermann tests confirmed the finding.

A full description is furnished of the employment of the instrument, and its simplicity and quickness of working are stressed as being of direct clinical value.

J. S. P.

A peculiar form of hereditary familial disease (Une forme particulièure de maladie héréitaire familiale).—MARC SREJSKI. *L'Encéphale*, 1928, xxiii, 821.

The cases here described are those of three brothers and one sister of the ages of 29, 22, 20 and 18 years who suffered from night-blindness, deafness, mental backwardness, speech defects (dysarthria and agrammatism) and a certain degree of sexual infantilism. Hearing, speech defects and mental retardation had been present since infancy. Night-blindness associated with retinitis pigmentosa came on in all four at the age of 15. The parents were Russians, non-Jewish, and were not related. They showed no stigmata of nervous disease. The author discusses the possible relationship of this condition to the juvenile type of amaurotic family idiocy.

J. G. G.