Lead poisoning is seen occasionally in infants and children from the prolonged use of lead nipple shields or from the ingestion of water containing lead; also lead is reported to have been obtained from face powder containing lead used by the child’s mother; however, the most common cause of the ingestion of lead appears to be the habit of small children of chewing paint from toys, cribs or woodwork of the house. If the ingestion of lead paint continues for any considerable time, symptoms of intoxication result. True perversion of the appetite, so-called pica, appears to develop in some children; in addition to paint, sand, coal, cloth or hair may be ingested. Mentally defective and highly neurotic children are prone to show evidences of pica; bizarre appetites may develop in anaemic children. and children harbouring certain intestinal parasites are notorious in this respect.

The early manifestations of lead poisoning are those referable to the gastro-intestinal tract—anaemia, constipation, and colic—associated in most cases with a variable degree of anaemia. More serious symptoms are those referable to the central nervous system—vomiting, frequently of a projectile character, visual disturbances, delirium, stupor, coma or convulsions. These neurological evidences of lead poisoning often are associated with an elevation of the arterial blood pressure, choking of the optic discs or even separation of the cranial sutures. If death occurs, it usually follows a period of coma or severe convulsions. While the heart continues to beat for some minutes, the respirations cease, apparently because of central respiratory involvement.

The cerebral manifestations of lead poisoning appear to be due to extreme cerebral oedema resulting from the deposition of lead in the brain. This marked cerebral oedema was observed by Chvostek in 1897, and has been produced experimentally in laboratory animals by Weller.

If the patient survives a severe encephalitic stage, permanent cerebral sequelæ may remain, among them blindness, cerebral palsy, cerebral atrophy and degeneration, internal hydrocephalus, epileptiform seizures and mental deficiency, all presumably the result of impaired circulation in the brain during the prolonged state of increased intracranial tension.

The development of encephalitic manifestations is more frequent in children than in adults, whereas the type of neurological involvement commonly seen in adults, peripheral neuritis, has been encountered relatively seldom in children.

The early diagnosis of lead poisoning based on a carefully taken history and physical examination, confirmed by röntgenological laboratory data, should lead to the institution of active therapy, which may result in the
prevention of the development of serious neurological manifestations. In the presence of the acute, severe cerebral manifestations, attention must be directed toward the control of greatly increased intracranial tension.

R. M. S.


The authors report a case showing certain arterial lesions in the central nervous system of an unusual type.

Clinically, there was a combination of pyramidal, extrapyramidal, and pseudobulbar signs, with an unusual manifestation of tonic innervation in the left hand. The course of the illness was more continuous and the dementia was deeper than that ordinarily observed in cerebral arteriosclerosis. The results of the serological tests were negative. The clinical picture was complicated by the presence of a paraplegia in flexion. This was found to be due to an encapsulated tumour of the cauda equina. The tumour was evidently an independent process, for it bore no apparent relation to the vascular changes.

The vascular changes were limited to the smaller arteries and arterioles of the central nervous system. The first alterations consisted in swelling and proliferation of the endothelial cells, whereby masses of large, rounded elements with translucent but slightly granular cytoplasm were formed. This resulted in complete or partial blocking of the lumina of the affected vessels. Following this, the newly formed cells underwent severe fatty degenerative changes, which often spread to the media. Complete disintegration of the walls of the vessels frequently ensued, with the occurrence of thrombosis and hemorrhages. The end stage of the process consisted in connective tissue organization of the involved vessels. There was no tendency to recanalization, and splitting of the elastica interna was not observed. Calcification did not take place.

The nature of the vascular disease would seem to be almost as obscure as its etiology. Histologically, there was a combination of productive and degenerative changes which, theoretically, might result from the association of arteriosclerosis and endarteritis syphilitica attacking the smaller cortical vessels. It is, however, very improbable that two independent disorders would merge with each other completely enough to produce alterations of a uniform character in practically all parts of the central nervous system.

The possibility of a hitherto undescribed type of disorder must be entertained.

R. M. S.
In the group of cases reported, eight presented definite evidence of fracture of the skull; in two it was suggestive; two of the cases exhibited subarachnoid bleeding following the initial trauma, but in the majority there was no evidence of either fracture of the skull or subarachnoid bleeding. There were nine cases of posttraumatic epilepsy; six showed objective signs of a residual focal lesion in the brain. In one case there was evidence of a gross lesion in the substance of the brain without subarachnoid bleeding or fracture of the skull. In a similar instance studied more recently at autopsy, a large intracerebral haemorrhage was found without signs of disease of the cerebral vessels. There was also no evidence of fracture or subarachnoid haemorrhage.

In all but one of the cases studied, definite changes in the encephalogram were demonstrable. These consisted of dilatation of the ventricles, considerable accumulations of air on the convexity of the brain and migration of the ventricular system toward the site of the lesion. These deviations from the normal were found either separately or in combination.

It is, of course, understood that no sweeping conclusions can be drawn from this relatively small group of cases. Yet the encephalographic observations were so uniformly abnormal that they are suggestive of an organic basis for some of the symptoms in the posttraumatic. In a number of the cases signs of focal disease were demonstrated, but even in those with only general symptoms a similar process probably forms the basis of the complaints.

The author concludes that encephalography offers a valuable means of differentiating between organic and functional syndromes.

R. M. S.

The pupillary phenomenon of Argyll Robertson is a complex one and no one of its components is more constant than another. The author asserts that the 'true' Argyll Robertson pupil is invariably associated with neurosyphilis and is never found apart from that condition. It commonly develops segmentally, becoming uniform only when fully developed. It is to this segmental development of iris changes that segmental loss of light reaction and pupil irregularity are due. This mode of development indicates that the underlying nervous lesion must be in the peripheral neurone. The observations made by the author are not compatible with a central lesion in
the midbrain. The Argyll Robertson pupil of non-syphilitic lesions of the nervous system is clinically distinguishable from the ‘true’ Argyll Robertson pupil. It is accompanied by pupil dilatation, and characterized by an absence of miosis, pupil irregularity or trophic changes in the iris. Theories as to its pathogenesis have no bearing upon the problems presented by the ‘true’ Argyll Robertson pupil.

C. S. R.


Five personal cases were studied post-mortem and several in the literature are discussed. The author concludes that early hypertonus manifested in man coincidently with the onset of cerebral haemorrhage, softening, etc., is an expression of lesions not in the pyramidal tracts or in fronto-pontine or temporo-pontine tracts, or in thalamus, but in the corpus striatum of the same side (neo- and paleostriatum) in the mesencephalon (substantia nigra, probably also in the substantia reticulata) and in the pons. If the corpus striatum is spared, hemiplegia from interruption of the pyramidal tract in one hemisphere is found without hypertonia; however in spite of the alteration in the striatum it is sometimes possible to find hypotonia, when the nervous system on the side of the lesion is transformed into a mesencephalic organism.

The Magnus-de-Kleyn reflexes in man result from lesions of lenticular paths in the peduncles of the pons, also perhaps from the descending tracts from this last area. Lesions of the pyramidal and other tracts are not necessarily found. Sometimes these lesions are absent in cases of complete 'mesencephalisation' or because the muscular rigidity is of another origin. The centre for the reflexes in man is situated in the fifth cervical segment.

R. G. G.


The author describes six cases illustrative of the difficulty of diagnosis of this form of neuritis. He remarks that had not chemical examination revealed the presence of large amounts of arsenic in the hair, the etiology and even the nature of the lesions in some of the cases here recorded would have been in doubt. Of equally great important was, in his opinion, the presence of transverse bands on the nails of the fingers and toes. According to Mees such bands are actual deposits of arsenic in the nails and their presence
alone, that is, without a chemical examination of the hair, should, in the writer's opinion, suffice for a diagnosis of arsenical poisoning. They should always be looked for, and their presence would indicate the advisability of a chemical examination of the hair. As the number of contributions on the subject is so far rather few, it is not possible to estimate the relative value of white bands in the nails and the presence or absence of arsenic in the hair. Some of the cases which have been and still are under his observation showed distinct bands in the absence of appreciable quantities of arsenic in the hair. In one case no arsenic was found in the hair at all though the nail bands were distinct, while the clinical picture of arsenical polyneuritis was quite clear.

It is evident that further observations of cases as outlined in this article should be recorded. It is noteworthy that in none of the author's cases was it possible to determine the source of the arsenic and that the clinical picture was mostly atypical, with prevalence of motor disturbances.

R. G. G.


A case of acute epidemic encephalitis followed by bulbar palsy and later a syndrome of amyotrophic lateral sclerosis is reported. A prominent feature of the case is universal myoclonia affecting all the muscles of the body. Lesions of the anterior horns and pyramidal tracts are inferred to explain the amyotrophic lateral sclerosis picture; lesions of the mesencephalon are thought to be the cause of the universal myoclonia rather than ganglion-cell irritation in the motor nuclei of cranial nerves and anterior spinal horns.

R. G. G.


A case is described in which as is usual in cases of syringomyelia complicated by choked disc the symptoms of intracranial pressure are so pronounced as to overshadow those of syringomyelia. The question of tumour always arises but in this case as in others the condition is due to hydrocephalus connected with closure of the foramina in the roof of the fourth ventricle.

R. G. G.

This family consisted of six living children, two of whom were normal, while four presented the combination of cataract, rickets, imbecility and infantilism which forms the title of the paper. No etiological factor could be discovered, and in particular both congenital syphilis and pellagra were definitely excluded. But the blood calcium was found to be well below normal not only in the affected children but also in the healthy children and the father. The authors consider that the cause of the disease was a disturbance of the endocrine balance with special default in the parathyroids. The cataract also seemed to be dependent on diminished parathyroid secretion, seeing that the combination of rickets with cataract is usually associated with tetany. Seeing that good results had occasionally been claimed for the treatment of imbecility with parathyroid extract, the authors subjected their patients to this as well as ultra-violet rays, but without obvious good effect. Injection treatment with parathyroid extract does not seem to have been tried.

J. G. G.

PROGNOSIS AND TREATMENT.


Dehydration was carried out on a series of typical institutional epileptics but had no definite effect on the occurrence of fits or on the patient’s disposition. The giving of unlimited fluids after a period of dehydration and the forcing of fluids without previous dehydration had no definite effect on the number of fits. The patients were adversely affected by the diet, and preliminary loss of weight was severe. Resistance, too, was lowered. Nitrogen retention either precipitated or complicated stages of excitement and stupor, and acidosis occurred during the excitements. The results obtained in 12 cases were such that this method would appear to be of little value. This conclusion is reinforced by the injurious effects noted.

C. S. R.

Endocrinology.


The author conducted previous experiments on birds and now repeats them in dogs. As a result of these experiments he finds corroboratation of his previous work, that pituitary lipoidal substance—the indication of activity of the gland—is in excess when sexual activity is in abeyance, and undergoes diminution during periods of sexual activity. This applies both