The phenomena of epilepsy are apparently responses on the part of neural mechanisms to various forms of noxious stimulation. The kind produced depends more on the kind of function of the neural mechanism which responds than on the nature of the stimulus that produces the response. It is hardly much of an exaggeration to say that the normal individual of 50 cannot have arrived at that age without some form of epileptic phenomena having occurred in his life. This is a conception which might be set against that held by a great many doctors that epilepsy is necessarily a progressive, practically incurable, and always deteriorating disease. One might speak of the epilepsies as though they were a spectrum—a spectrum the colours of which blend the one into the other. In that spectrum is the great fit, following an ordered pattern of sudden unconsciousness and decerebrate rigidity, followed, apparently due to mesencephalic block, by the disordered resumption of control by the cortex, followed by exhaustion, followed by resuscitation. In that same spectrum are many other forms of seizure. By giving them special names we have obscured our own consciousness that eclampsia is epilepsy, that uremia is epilepsy; and we find the epileptic effects of insulin changing the content of blood sugar as alterations in the metabolic rate of the brain. It is almost as if the brain were in a state of awareness against anything that would interfere with the proper oxygenation of the cell. Any form of deoxygenation of the cell increases the irritability of the cell and the energy discharge is a response to anoxemia. This is true as seen clinically in the epilepsy of the alcoholic brain. It is true of the brain of the paralytic. It is true of any increased water content of the brain, that the film of fluid interferes with the oxygenation of the cell and permits or precipitates the discharge of its energy. It is possibly true in another domain—an urgent response by neural centres to irritants producing localized brain oedema. Localized collections of fluid can produce such states of deoxygenation of cells as to produce an epileptic discharge. Undoubtedly, too, the vasovagal attacks of Gowers are discharges along sympathetic paths. We have, as it were, rediscovered the epilepsies of the sympathetic system. Further, the psychic equivalents are undoubtedly an expression of disordered integration of the brain—appearing as sensory or motor phenomena. The psychic
phenomena of epilepsy should suggest the somatic origin of many of the psychoses.

C. S. R.


Two cases are reported in which autonomic manifestations (nausea, epigastric distress and vomiting) were often substituted for epileptic convulsions. From a study of these cases it would seem that both sensory and motor autonomic representation exists in the cerebral cortex of man, but exact localization is impossible in the present state of knowledge. Epigastric auras and other visceral sensations described by patients are not so-called referred pains, but the result of vigorous and perhaps abnormal movements of the gastrointestinal tract.

R. G. G.


The author describes as an epileptic equivalent a rare symptom which appeared in an epileptic subject. This was a recurring pavor nocturnus with a consequent motor reaction of unconscious flight. This was repeated during sleep whether by day or night and was intimately related to the fundamental epileptic state.

The author discusses emotional reactions during sleep in epileptics and their relationship to the actual epileptic attack.

R. G. G.


The case of a mentally retarded three-year-old child who showed the unusual symptom of flexibilitas cerea is here presented. Following a period of observation, the catalepsy gradually disappeared simultaneously with a progressive improvement in the intellectual status. It is suggested that this type of catalepsy is physiological or developmental in nature. The relationship to the general question of catalepsy is discussed.

C. S. R.


Little’s disease, i.e. congenital spastic quadriplegia, shows at autopsy sometimes signs of cerebral alteration, sometimes spinal (agenesis of the
pyramidal tract); sometimes both parts are affected. In cases of primary spinal lesions the patients usually retain a normal intelligence, have normal facies and do not show squint or epilepsy. In other cases afflicted by more or less serious defect the lesions are generally found in the brain. In the present case a status verrucosus deformis in the brain was discovered. Hypoplasia of the medulla existed but was not sufficient to account for the gravity of the symptoms by itself; they seemed to be secondary to the destruction of cortical pyramidal cells. The case is similar to that described by Pellizzi, that of a patient with generalized spasm, epileptic convulsions, and idiocy, who was born at the seventh month. At post-mortem examination the brain showed microgyria in the neighbouring convolutions and arrested development of the pyramidal tracts.

With regard to the pathogenesis of the verrucous condition Ranke has demonstrated that towards the fourth and fifth month of foetal development before the appearance of the convolutions, an atrophic condition of the cortex might develop in special circumstances, assume a pathological appearance, and spread more or less on the surface, but also extend from the surface into the interior of the cortex. Ranke believes that such deformity is not always dependent on defects of development, but can be provoked by morbid processes. Kraepelin states that such cases are as a rule idiots with epilepsy. In the present case besides the idiocy and rare epileptic attacks there also existed a congenital quadriplegia. Bourneville stated that such lesions resemble those of tuberose sclerosis or foetal encephalitis. It is probable that here the condition was provoked by a foetal meningoencephalitis at the fourth or fifth month which began in the cortical zone and from there extended more or less to the whole brain. In this way may be explained the adherence of the meninges, the thickening of the vessels, the accumulation of neuroglia, the disordered cortical architecture and the presence of pathological cells. The aetiology of such foetal meningoencephalitis is unknown.

R. G. G.


The writers studied 106 consecutive cases of clinical uncomplicated mastoiditis without intracranial extension and found that already many neurological signs were present. They discovered alteration in normal reflexes, mild eyeground changes, pathological reflexes, focal headaches, pupillary changes, pain in the eye of the same side, vomiting or severe nausea, nystagmus and meningeal signs in that order of frequency. They do not find in their series of cases any aphasia, cerebellar symptoms, extraocular palsies, perimetric changes, focal convulsions or pareses.

R. G. G.