The limpidity, tension and Wassermann reaction in all cases were normal. In two-thirds of the cases there was slight increase (± to 3+)

of the globulin content (method of estimating not stated). The cell count per c.mm. varied from 0 to 10, i.e., was normal. In every case, except one in which the nervous symptoms were minimal, there was a positive colloidal gold reaction. This occurred in the middle zone (luetie), and was slight, rarely reaching 3. It occasionally extended into the higher dilutions. Both globulin and gold reaction became normal as the paralysis disappeared.

M. A. Blandy.

SENSORIMOTOR NEUROLOGY.


A. Many observers have previously called attention to disturbances of the respiratory function in this disease (reference is made to their papers). Symptoms of this nature may be classified as:—

(1) Respiratory disturbances properly so-called. These are: apnoea occurring either continuously or in nocturnal paroxysms; disturbances of the normal rhythm in the form of occasional or periodic phases of apnoea; and sighing.

(2) Paroxysmal coughing without expectoration.

(3) Respiratory ties (sniffing, blowing and spitting) and abnormal sensations in the nose and larynx.

B. Lethargy is far from being the only disturbance of the normal sleep-rhythm. Insomnia is often encountered in various forms. Invasion of the normal cycle of sleep and wakefulness, retardation of the sleep hour, restless sleep, somnambulism, periodic excitement at fixed hours in the evening, are described in detail.

A series of cases is briefly recorded to illustrate the two groups of symptoms. The paper, which is rich in clinical detail, should be consulted in the original by those interested in the subject.

C. P. S.


A woman of thirty-two suffered in February, 1920, from a febrile illness diagnosed at the time as influenza. Six months later she noticed an abnormal tendency to muscular fatigue and slowness of movement. Towards the beginning of 1922 her speech became embarrassed at times on account of stiffness of the lips and tongue. Examined in 1922, she showed general muscular rigidity and slowness of movement of the Parkinsonian type.

The most remarkable feature, however, was the contracture of the tongue, which at rest took the form of a ball held between the teeth. The
patient was nevertheless able to move the organ at will and could eat freely. She found difficulty in articulation, especially in the early morning. The mental condition was that which the author considers typical of the post-encephalitic state, apathy and slowing of intellectual effort with preservation of automatic activity and response.

C. P. S.

[73] Complete paralysis of the upper part of the face in hemiplegia from an extensive unilateral lesion of the cerebral cortex (Sur la paralysie totale du facial supérieur dans l'hémiplégie par destruction étendue unilatérale de l'écorce cérébrale).—VEDEL, G. GIRAUD and P. SMIREON. Revue neurol., 1922, xxxviii, 1270.

Involvement of the upper part of the face in hemiplegia, though unusual, has been recorded in a number of cases. The anatomical basis for the more usual escape of these muscles is uncertain. Two hypotheses are mentioned:

1. That the upper neuron supply is bilateral; 2. that the cortical centre for the upper face lies in the angular gyrus with a centrifugal pathway distinct from the pyramidal tract.

The case is recorded of a man of forty-seven, afflicted with renal disease, who suffered from an ictus with complete left hemiplegia. The whole of the left face was involved. Ptosis and a fixed dilated pupil indicated also a lesion of the left third nerve. The autopsy revealed an extensive softening of the right hemisphere, due to a thrombus involving the anterior and middle cerebral arteries and extending back to the internal carotid. The left hemisphere was intact (there is an unfortunate printer's error in the original). In addition a small hemorrhage was found involving the left third nerve at its point of emergence.

In discussing this case the authors quote others in which complete involvement of the face was associated with sudden extensive ischaemia of one hemisphere, as in wounds of the internal carotid in the neck. They take this as evidence in support of the hypothesis that the centres for innervation of the upper and lower halves of the face are separately situated. If the lesion is sufficiently extensive it will involve both centres, or their pathways. In relation to the ipsilateral lesion of the third nerve they do not refer to the hypothesis long ago put forward by Mendel that the orbicular and frontal muscles are supplied from the nucleus of the third nerve on the same side.

In the light of this hypothesis their case might be capable of another interpretation.

C. P. S.


This paper concerns a patient who, after having suffered from myositis in 1908 and again in 1914, died in 1918 with pronounced muscular atrophy. The attack of myositis in 1914 was severe, lasting three months and causing much pain and swelling in the muscles of the thighs. By May, 1915, the patient was well enough to enlist in the army, and it was not till a year later
that the first symptoms of muscular atrophy came on. Examination two
months after that revealed extensive wasting of the muscles of the shoulder
girdle and a moderate amount of wasting of the muscles of the left thigh.

His illness ran the course of a progressive muscular atrophy and he died
in January, 1918.

Post-mortem, no changes were found in the brain and spinal cord; in the
muscles there was extensive atrophy of the fibres; the remaining fibres were
very unequal in size, small atrophic ones and large hypertrophic ones being
found side by side; the spaces between the fibres were much greater than
normally; nuclei, greatly swollen and much increased in number, lay
together in rows among the fibres; the intramuscular nerve endings seemed
healthy; the interstitial tissue was in places infiltrated with lymphocytes.
In the femoral nerve and in the large trunks of the brachial plexus a few
degenerated nerve fibres were found, and on that account the author prefers
classing his case as one of neuromyositis to one of polymyositis.

J. P. M.

[75] Huntington’s chorea and progressive familial myoclonus-epilepsy
(Uber Huntingtonische Krankheit und fortschreitende familiäre
Myoclonusepilepsie).—SCHULTZE. Deut. Zeit. f. Nervenl., 1922,
lxxv, 319.

SCHULTZE here revives a former controversy as to whether or not Hunting-
ton’s chorea and familial myoclonus-epilepsy are different diseases or variants
of the same disease. Oppenheim and Lundborg took the former view, the
author and some others the latter, and so the major part of this paper is
devoted to a critical examination of, and an attempt to annihilate, the points
of difference between the two conditions. Common to both diseases are the
frequency of their familial occurrence, the presence of involuntary twitchings,
the frequency of progressive mental defect and a steadily progressive course.
But in Huntington’s chorea (1) direct inheritance is more usual; (2) the
onset is much later; (3) the movements are more comprehensive; (4) the
twitchings have more tendency to cease during voluntary movements;
(5) the gait is much less interfered with; and (6) epileptic attacks are very
much less common than in myoclonus-epilepsy. Schultze discusses these
differences one by one and shows that none of them is absolute. Patho-
logically neither condition is at all clearly defined, so that, although the
author devotes some space to them, arguments from pathology have little
or no weight.

At the end of the paper reference is made to some other cases of twitching
movements and to the so-called ‘ canine chorea,’ but no suggestion is ventured
as to their specific pathological basis.

J. P. M.

[76] The ecology of epilepsy.—C. B. DAVENPORT. Arch. of Neurol. and
Psychiat., 1923, ix, 554.

The aim of this paper is to inquire into the distribution in the human species
of the tendency to epilepsy. The author gives statistics (the majority of
which are taken from Army Reports), with twenty-eight references, concerning
the prevalence of epilepsy in the following groups: Negroes, Amerindians, Mongolians, East Indians, Philippine Islands, Polynesia, Europe, Great Britain, United States and European nations represented in it.

He concludes that the tendency for muscles, singly or in groups, to undergo tetanic contractions, more or less periodically without obvious cause, is widespread among higher vertebrates. Consequently its causative factors must be conceived broadly enough to include mammals and birds. Hence diet, alcohol, focal infection and trauma may be, and doubtless are, causative factors in particular cases, yet no one of them is the essential factor. The factor that is most nearly universally present is some inherited constitutional peculiarity of the body, so that the tendency for muscles to go spontaneously into tetanus recurs with especial frequency in certain families. It is à priori probable that certain strains or races differ in the rate incidence of epilepsy by virtue of either an inherited constitution or a difference in the incidence of one or more exogenous factors, or by virtue of both these causes. In-breeding is a danger in a population where the endogenous factors exist. In general, the statistics indicate that alcohol may be an important exogenous factor and that low rates of incidence are attributable to favourable factors, both endogenous and exogenous.

E. B. G. R.

[77] The pathogenesis of epilepsy.—Michael Osnat. Arch. of Neurol. and Psychiat., 1923, ix, 488.

The author reviews the problem of the causation of epilepsy in the light of modern research into the factors underlying the convulsions.

He first considers the possibility of a psychogenic origin, expressing little sympathy with the views of Pierce Clark. The fact that the delirium of ‘petit mal’ attacks deals with the content of consciousness does not prove a psychogenic origin, since all deliria are similar in this respect. Moreover, epileptic characteristics are possessed by many persons who do not suffer from seizures, and the theory of psychogenic origin cannot be applied to cases with a known organic basis. He hesitates, however, to discard this theory completely because of the work of Crile and Cannon. He invokes the psychogenic factor to account for epilepsy developing under the strain of warfare in a patient operated on twenty years previously for depressed fracture.

The mechanical and vascular theories of pathogenesis, as summarised by Sargent, find more favour. In this case the tonic stage of the fit, which is considered analogous to decerebrate rigidity, is explained by the cutting off of the cerebral circulation, and the clonic stage by the gradual, inefficient resumption of the same. Pike’s experiments on cats support this view. Sargent attributes these circulatory disturbances to alterations in autonomic control, but such control of cortical vessels is not yet proved. His theory does not, moreover, seem adequate to account for the development of epilepsy in only 4·5 per cent. of the 18,000 cases of cerebro-cranial wounds reviewed by him.

The observations of Krause, Sherrington, Sargent, Holmes, and others on individuals suffering from Jacksonian epilepsy are briefly referred to and the interpretation of the epileptic fit as a symbolically purposeful act is eliminated
on the ground that the movements produced by faradice stimulation of the cortex are exactly similar to those of the epileptic convulsion and non-purposeful in nature.

Cuneo's work is reviewed at length and his conclusions taken as the most promising basis for further research. Cuneo believes that albumosæmia exists in epilepsy. This is caused by an insufficiency of the alkali-forming function in liver and small intestine, by means of which organic salts, which are transformable into sodium carbonate, do not undergo this metabolic change, but enter the circulation and cause a division, under certain circumstances, of the nucleo-histose element of the nucleo-proteids into nucleinic acid and the proteoses. These latter remain free and exhibit their convulsive action. His conclusions are based on: (1) Investigations on the blood and urine of epileptics after seizures; (2) the effect on epileptics of a predominantly protein or predominantly carbohydrate diet; (3) studies, in vitro, of the action of intestinal mucosa, combined with liver or pancreatic tissue or bile, on the acid organic salts formed in metabolism of starchy substances; (4) production of nucleinic acid and proteoses by hydrolysis and oxidation of blood serum; (5) development of epileptic convulsions in animals by intravenous injection of the substances thus produced; (6) comparison of the post-mortem findings on these animals with those found in human beings dying in status epilepticus; (7) clinical observations that restricted carbohydrate diet and administration of alkali by mouth give encouraging results in epilepsy.

Reference is also made to Alzheimer's neurohistopathological findings in epileptic dementia. Bacteriological causation of epilepsy is dismissed as disproved, and the importance of heredity emphasized only in the case of mental defectives and paralytic idiots. In conclusion the author states that the factor of importance in epilepsy is the acidosis, regardless whether this depends on a viciously functioning carbohydrate metabolism, which causes a general toxicosis, or upon the local production of the toxic substance from disintegrating cellular structures, secondary to vascular disturbances. The effect is the same; the only difference is the extent of the convulsive reaction.

E. B. G. RIVINGTON.


The first of these papers represents an attempt to arrive at a more exact localization of the areas involved in the production of hyperkinetic movements. The opening pages contain a short review of the work of C. Vogt, Wilson, Hunt, and other authors. One case of the author's is described in
The principal clinical features of the case were: constant involuntary movements in muscles of facial and bulbar innervation and distal muscles of extremities; combination of typical athetotic movements with associated movements: preponderance of hyperkinetic over paralytic symptoms.

The results of the microscopical examination are fully described; changes in the cortex were confined to the anterior central convolution on both sides, and consisted of a diminution in number and size of the cells of the superficial layers. Similar changes were found in the corpus striatum, being evenly distributed in the globus pallidus and the putamen-caudate. The thalamus and cerebellum were normal.

The case is considered to fall into the group described by Wilson, Vogt, and Ramsay Hunt. The correlation of individual symptoms of the case with details of the morbid anatomy is fully discussed, and stress is laid on the absence of the changes found by Bonhöffer in the cerebello-thalamic system.

In the second paper Thomas describes a series of five cases, of which involuntary movements were the common feature. Four of these were cases of typical athetosis, associated with pyramidal lesions; in one case the movements were choreiform and myoclonic, and the pyramidal tract was not involved.

The author discusses at some length the extent to which the symptoms shown by these patients—athetosis, spasmus mobilis, chorea—may be attributed to disturbances of extrapyramidal tracts. He considers that there is no real significance in Lewandowsky's distinction between 'athetose double' and 'bilateral hemiahthetosis,' and prefers to classify such cases on purely anatomical considerations.

Considerable attention is given in both papers to the significance of associated movements, and the extent to which they may be independent of the accompanying pyramidal lesions.

N. Hobhouse.


A CLINICAL account with x-ray photographs of a case of this rare condition. The patient, a male, aged fifty-one, first noticed progressive enlargement of the head at the age of eighteen. Subsequently the malar prominences became hypertrophied. No other bones in the body were affected by the disease. At the time of presentation the head measured 66 cm. in circumference. The surface of the hypertrophied bones was perfectly smooth to the touch, but the x-ray showed an irregular, mottled appearance, indicating that the density of the new bone formation was by no means uniform. The thickness of the cranial vault in places attained 2½ cm.

The authors seek to distinguish this condition from Paget's disease because (1) it was confined to the bones of the skull, (2) the appearance of the bones in the x-ray photographs was more spongy than they have seen in that disease.
ABSTRACTS

In searching for other examples of the condition which they describe they have found recorded as leontiasis ossea specimens showing nodular hypertrophy of the facial bones which they believe to be of syphilitic origin. The point of distinction is the smoothness of the surface in true leontiasis ossea.

C. P. S.

PROGNOSIS AND TREATMENT.


The author comes to the following conclusions:

1. General paresis is an active spirochetal disorder which, theoretically, can be arrested.

2. Despite a great divergence of opinion as expressed in the literature, there is considerable evidence that long-continued and intensively given antiluetic treatment will arrest the paretic process in a reasonable percentage of cases, especially if begun early in the disease.

3. In the Boston Psychopathic Hospital the experience has been that antisyphilitic treatment is of considerable value in cases of general paresis and that many paretics, even after the onset of marked psychotic symptoms, may recover sufficiently to resume their former occupations for a period of years, in some cases for more than seven years, without a return of symptoms.

4. Remissions may result from the use of intensive intraspinal or intraventricular injections when these apparently could not be obtained from intensive intravenous treatment alone.

5. More potent antisyphilitic drugs and more efficient methods of penetration of the drug into the tissues of the central nervous system may be expected to give better results in cases of general paresis.

6. Non-specific therapy, especially the inoculation of paretics with malaria, is reported in the German literature to give very satisfactory clinical results. It is necessary to keep an open mind at present on the value of non-specific therapy and methods of increasing the patient’s immunity.

7. There is reason to believe, both from the reports in the literature and from personal experience, that the paretic process may be arrested, symptomatology halted, and the patients returned to social activities and productive work.

C. S. R.


General paresis became established as a disease entity with a characteristic pathology in 1904, as a result of the work of Nissl and Alzheimer. As Alzheimer points out, there is rarely any difficulty in differentiating paresis from non-syphilitic brain disorders. However, in the case of syphilitic brain disease the differentiation is by no means so easy. The distinction between chronic tertiary syphilitic meningo-encephalitis and general paresis is at times very difficult, if not impossible. It would seem as if the line of demarcation between these two conditions is one of degree, and that at times the one disorder merges into the other. A pathological study of brains from cases