findings in the cerebrospinal fluid. In addition to purely vascular syphilis, which only gives positive findings in the fluid if there is marked inflammatory reaction round the vessels, negative findings may occur in tabes. Cerebral gumma, syphilitic cerebral nerve palsies, Erb's spastic paralysis, paraplegia, epilepsy, paranoia, and dementia. They regard the Argyll Robertson pupil as being pathognomonic of syphilis, and consider that the experienced neurologist may diagnose neurosyphilis from various characteristic combinations of symptoms in the absence of laboratory findings. The general opinion is that positive findings in the fluid exist for many years before symptoms are manifested, but exceptions may occur, and some believe that syphilis confined to the brain always gives negative fluid findings. It has been shown that the gold reaction varies in the same patient according as the fluid is taken from the ventricle, the cisterna magna, or the lumbar sac.

Cases of tabes with negative findings include (1) cases of incipient progressive type, (2) cases in which pathological findings have disappeared after treatment, (3) abortive tabes. Case notes are given of all types of neurosyphilis with negative fluid findings.

R. G. GORDON.

SYMPTOMATOLOGY.

[8] Two cases of cerebrospinal cysticercus infection with chronic meningitis and endarteritis of the brain (Deux cas de cysticercose céphéro-spiinaie avec méningite chronique et endarterite oblitérante céphalique).—L. REDALIE. Revue neurol., 1921, xxxvii, 241.

The two cases may be summarized as follows. The first was that of a woman, age 54, with a nine months' history of headaches and pains in the back, and occasional attacks of vertigo without loss of consciousness. Some weeks before death there developed retention of urine, constipation, paralysis of the limbs, paresis and dilatation of the left pupil, disturbances of sensibility, exaggeration of the deep reflexes, and extensor plantar responses. The intellect failed, there was swelling of the optic discs and a positive Kernig's sign, and finally the patient died with bed-sores and cystitis. Lumbar puncture some weeks before death showed an increase of albumin, 210 cells per c.mm., and a positive Wassermann reaction.

At the autopsy there appeared to be a considerable degree of internal hydrocephalus and a chronic leptomenigitis affecting mainly the base of the brain and the cervical region of the cord. Histological examination showed structures resembling the cysts of cysticercus in the meninges of the ventral surfaces of the pons and of the cervical cord, and an oblitative endarteritis of the large and medium-sized vessels at the base of the brain.

The second case was that of a man, age 66, who had suffered for fifteen years from epilepsy and for some months had noticed difficulty in walking. He was admitted to hospital with a diagnosis of multiple cerebral thrombosis. The Wassermann was negative in the blood, positive in the spinal fluid; the latter contained at that time 33 cells per c.mm.
He was treated with salvarsan and mercury, and two months later the Wassermann reaction was negative in the spinal fluid, which, however, still contained 83 cells per c.mm. Clinically, the patient made no improvement, and died somewhat suddenly a few months afterwards with signs of medullary failure.

In this case at the autopsy several cysts of different sizes were apparent upon the surface of the brain protruding from the subarachnoid into the subdural space. The largest of these measured nearly 5 cm. in diameter. They were most numerous at the base and in the lips of the Sylvian fissures. In addition there were chronic basal meningitis and endarteritis of the basal vessels. These latter changes were especially well marked in the basilar artery, the intima being enormously thickened, the internal elastic lamina broken, the media degenerated, and the adventitia converted into a mass of granulation tissue. On section of the brain, cysts were found in the cortex of both frontal lobes, and a single specimen the size of a haricot bean was discovered in the third ventricle. The spinal meninges were not seriously affected, but a cyst 1 cm. in diameter was visible on the lateral surface of the cord in the dorsal region.

In his discussion of these cases the author devotes much space to a detailed account of the histological appearances, especially those in the vessels. These, together with the diffuse thickening of the leptomeninges gave rise to a picture resembling that produced by syphilis, and this was all the more striking in view of the positive Wassermann reaction in the spinal fluid in both cases. Commenting upon the latter finding, the author takes it to mean that there was in both cases a coincidence of the two infections, spirochaete and cysticercus; but he quotes other cases from his own observations to show that the Wassermann may be positive in the spinal fluid in the absence of clinical evidence of syphilis.

It is noted that in neither case was the adult form of cysticercus found in the intestine; the ova therefore must have been ingested as such or have outstayed the parents in the host.

There are many references to the literature, and the paper should be consulted in the original by all who are interested in the subject.

C. P. Symonds.


The cases of three patients are presented, one of whom was improving, one was dead, and one was in statu quo. These cases presented clinical pictures simulating myasthenia gravis so closely that the positive diagnosis was not definitely made for some time. The apparently acute onset of the illness, the age of the patients in Cases 1 and 2 at the time of onset; the lack of variations in the degree of the muscular weakness; the absence of the true myasthenic fatigue phenomenon, and of the myasthenic electrical reaction; the persistent hyperactive inexhaustible deep reflexes; and the complete persistent external ophthalmoplegia, pointed against the diagnosis of myasthenia gravis. The persistent eye-muscle palsies, the unequal pupils in Case 2, the increase in the deep reflexes in all three patients, the bladder
disturbance in Case 2, the tendency to the Babinski phenomenon in the first two patients, the atrophy of the muscles in all three patients, the acute course of the disease in Case 2, and the lack of remissions formed the basis for the diagnosis of acute epidemic encephalitis. The pathological findings in Case 2 confirmed the diagnosis in one case in a group of three patients who presented remarkably similar clinical pictures.

R. G. Gordon.


The course and symptomatology of these cases is dealt with fully; but the most interesting part of the paper deals with sequelae, which are thus compared with sequelae in adults:

Weakness or paralysis: Adults, 8; children, 4.
Tremor, choreiform movements, spasm: Adults, 6; children, 3.
Pains in body or limbs: Adults, 2.
Headache: Adults, 8.
Dizziness: Adults, 3.
Nervousness: Adults, 5; children, 6.
Change in disposition: Adults, 1; children, 3.
Insomnia: Adults, 3; children, 2.
Drowsiness: Adults, 1; children, 3.
Speech defects: Adults, 6; children, 3.
Defects of eyes: Adults, 9; children, 4.
Change in mentality: Adults, 14; children, 10.

The changes in mentality are of great interest. In children there are usually indications of mental deterioration—the child is ‘demoted’ in school when previously it had made good progress. In adults there is loss of memory and of ability to concentrate, in the milder cases, and sometimes mental depression. In one instance a young woman committed suicide during convalescence. One man was at a hospital for the insane for some months, but was finally paroled. A woman had been at such an institution for over a year.

It is interesting to compare these frequent, diverse, and serious sequelae with those of poliomyelitis and epidemic meningitis. In the former, the sequelae are certainly sufficiently grave, but they are confined almost entirely to flaccid paralyses. In meningitis only a very small percentage of sequelae occurs. The most serious is deafness, and this occurs in only 3 or 4 per cent of cases. Blindness, due practically always to a panophthalmitis, is still more rare. While a popular opinion exists that mental defects are often due to meningitis, a careful study of recovery cases of meningitis fails to corroborate this opinion.

Relapses after comparatively short periods of improvement occasionally occur. In one instance, already referred to, there was a second attack, at the expiration of a year of good health, which proved fatal.

R. G. Gordon.

This somewhat discursive but informative paper is based on the study of some 36 cases of cerebral tumour personally observed. Among the matters of neurological interest are the following.

1. In several cases the presence of nystagmus could not be set down either to local or neighbourhood lesions, and is ascribed by the author to the rise of intracranial pressure producing a 'labyrinthödem' analogous to papillödem (Stauungslabyrinth, Stauungspapille). The vertigo of many cases of intracranial tumour, similarly, is not of localizing significance, or but rarely so.

2. In four cases disturbance of bladder function was observed (retention, precipitate micturition), in two and perhaps three of which the lesion was presumably bilateral; but they do not lend definite support to the view which ascribes sympathetic control to 'centres' in the basal ganglia (Czyhlarz-Marburg and others).

3. In 20 out of 28 cases, examination by x rays revealed recognizable changes: (a) Calcification foci within the brain; (b) Alterations in the sella turcica; (c) Changes in the skull produced by rise of pressure; (d) Local changes in the cranial bones. It appears, however, that none of these is of much localizing usefulness; thus in no less than 13 cases the sella turcica and its vicinity were altered, yet the author specifically states that in none was the lesion local.

4. One very interesting case of frontal tumour is given in detail, since the patient showed in characteristic form not only motor apraxia but the phenomenon of tonic innervation. At the operation a tumour the size of 'a man's fist' was found occupying the first and second left frontal gyri. The symptom of tonic innervation (also called, less accurately, tonic perseveration) is discussed at some length, and ascribed to a lesion of frontocerebellar paths; but there is no reference to the striking case of Goldstein in this connection, or to the contribution on this subject made by Wilson and Walshe.

S. A. K. W.


The author points out that once a diagnosis of tabes with gastric crises is made, all thought of ulcer is usually dismissed, but he describes three cases. The first was one of violent tabetic crises with symptoms very suggestive of gastric ulcer, but one in which the existence of the latter complication could not be established. The second was a case of tabes with predominant gastric symptoms and an apparent duodenal ulcer. The third was an example of advanced tabes with vomiting, hematemesis, and melena due to an active bleeding duodenal ulcer.

The frequency with which ulcer co-exists with tabes cannot be stated; that it can so co-exist is certain. The pathogenesis of the ulcer as a complication of, or coincident of, tabes is probably as follows: cerebro-
spinal syphilis is accompanied in a large percentage of cases by gastric hypersecretion. Organic lesions of the spinal cord or brain often cause delayed gastric motility, and probably abnormal gastric peristalsis. These two conditions presumably predispose to gastric or duodenal ulcer. Syphilitic arteritis may also play a rôle. Such ulcers as form are probably simple peptic and not syphilitic ulcers or syphilis of the stomach. The point of origin of the secretory and motor disturbances in the stomach and intestine is probably in the involvement in the pathologic process of the sympathetic fibres to these viscera in their passage through the dorsal spinal ganglions and posterior nerve roots. It is quite possible that the finding of a gastric or duodenal ulcer in tabes is a pure coincidence, and that there is no relationship of cause and effect between the two conditions.

R. G. GORDON.

TREATMENT.


In this somewhat dogmatic paper the author castigates modern brain surgery, and concludes that: (1) Brain tumours are among the most frequent neoplastic lesions; their growth is always progressive, and almost always leads to a train of terrible sequelæ and eventually to death. (2) There is only one form of treatment for tumours of the brain—operative removal—and this must be complete. (3) To obtain the best operative results, brain tumours must be diagnosed and localized in the earliest stages. (4) It is now possible to diagnose and localize practically every tumour, and in the early stages. When all other signs and symptoms fail in the localization, cerebral pneumography will make the diagnosis and localization with precision and without equivocation. And when a tumour is not present, it can be excluded by the same method. (By cerebral pneumography the author means x-ray photography after inflating the ventricles or spinal canal, or both, with air. This procedure, he points out, is only safe or justifiable when performed by skilled operators; but he claims that by alterations in the spaces so demonstrated the presence and localization of a tumour can almost infallibly be demonstrated.) (5) The operative approach will be dictated by the precise localization. The approach should afford adequate room, and it should be directly over the tumour. (6) After correct localization, all brain tumours should be diseased at operation. (7) Every effort should be made to cure the patient by complete extirpation of the growth. There is less mortality from carefully performed tumour extirpations than from unsuccessful explorations for tumours. When, for any reason, it is impossible or unjustifiable to remove the tumour, the maximum palliative relief should be given at the same operation. (8) Decompressions, ‘routinely’ performed, are among the most harmful and indefensible operations in surgery. They should never be performed for unlocalizable tumours. They are the exact equivalent of giving morphine for abdominal pain; the symptoms are masked until it is too late. (9) Decompressions should be performed only as a last resort—when the tumour cannot be removed—and then only after the location of the tumour.