PATHOLOGY.


General acceptance of the obstructive theory of hydrocephalus has for long been prejudiced by the impressive argument that in most cases there is no apparent obstruction or, indeed, cause of any kind. In the present contribution Dandy advances experimental evidence in proof of his contention that the commonest cause of all types of internal hydrocephalus is diminished absorption of cerebrospinal fluid, following obstruction of its outlet from the cerebral ventricles or subarachnoid space.

In one series of experiments a small piece of cotton enclosed in an oiled gelatin capsule was introduced into the aqueduct of Sylvius. Dilatation of the lateral and third ventricles resulted. The dogs on whom this procedure was performed became lethargic, and suffered from intermittent vomiting. Enlargement of the head was not noted, as the animals were of an age when the sutures are closed. It was also found possible to produce a unilateral hydrocephalus by blocking the foramen of Monro with a piece of transplanted fascia or peritoneum. If at the same time the entire choroid plexus was removed, the ventricle, instead of enlarging, became obliterated, thus affording conclusive proof that cerebrospinal fluid is formed by the choroid plexus, and that the ependyma lining the ventricles is not concerned in its production.

In another smaller series of experiments the Sylvian aqueduct was occluded after extirpation of the choroid plexus from both lateral ventricles. This was followed by a slowly-developed hydrocephalus owing to the production of fluid by the intact plexus of the third ventricle. Ligation of the vena magna Galeni close to its origin gave rise to ventricular dilatation; but in this type of experimental hydrocephalus the aqueduct of Sylvius and the foramina of Monro, Magendie, and Luschka all become enlarged, because the accumulation of fluid is due to over-production, there being no impediment to localize the dilatation.

While the appearances produced experimentally were very similar to those seen in the idiopathic hydrocephalus of children, Dandy considers vascular occlusion a rare cause of hydrocephalus with communication. Such cases are far more commonly due to adhesions which obliterate the basal cisternæ. To disclose adhesions and obliteration of the subarachnoid space, the brain must be studied during its removal; subsequent examination of the brain reveals but little, as the adhesions are liberated by removal of the brain, and the gross appearance of thickened pia-arachnoid is not striking after fixation.

The proof of Dandy's explanation lies in the ability to reproduce communicating hydrocephalus in animals. When the midbrain of a dog is enclosed by a strip of gauze which has been saturated with an irritant, adhesions form, and, acting as a barrier, they prevent the passage of cerebrospinal fluid to the subarachnoid spaces over the hemispheres. Consequently, the area in which absorption of cerebrospinal fluid can occur is limited to about one-fifth of the normal amount, and dilatation of the
ventricles results. In reality, therefore, both forms of hydrocephalus have the same origin and differ only in the locality of the obstruction. R. M. S.

[34] A note on the pathology of the choroid plexus in general paralysis.—A. E. Taft. Arch. of Neurol. and Psychiat., 1922, vii, 177.

In sections of the choroid plexus from cases of general paralysis, Taft found a progressive fibrous change, beginning with general increase of connective tissue, followed by obliteration of capillaries with formation of fibrous tufts, in which calcium salts are deposited. Finally, the capillaries disappear and the plexus becomes cystic, but the ependymal cells remain and are little changed morphologically. In view of these findings, and of the abundance of cerebrospinal fluid in this disease, Taft asks whether we are justified in concluding that the persisting ependymal cells are capable of functioning in the rôle of gland cells without the presence of the capillaries with which they normally stand in relation. R. M. S.


From two specimens which he has studied, the author of this paper deduces that certain cases of anencephaly are due to an inflammatory process in the embryo. In the first specimen he found, in both the grey and white matter of the cord, dilated vessels and hemorrhages, the latter almost blotting out the normal picture of the cord in section. In many places collections of small round cells occurred, especially towards the periphery; the cells seemed to have passed inwards from the meninges, which were infiltrated and thickened. The most marked changes were found in the highest part of the nervous system, where the central canal opened out and there was a flat continuation of the cord. This part contained absolutely no ganglion cells, so it could not definitely be called medulla; much of it consisted of undifferentiated tissue, in which hemorrhages and small round cells abounded. The second specimen showed essentially the same features, but the medulla was distinguishable and the twelfth nucleus was present.

It is interesting that in the first case, where no medullary nuclei were present, the child breathed for fifteen minutes after birth—a fact from which Pekelsky deduces that the respiratory ‘centre’ does not exist as an anatomical entity.

J. P. Martin.


Fuchs, in 1913, produced choreiform movements in cats by guanadin, and the animals died of the intoxication and showed post mortem a general inflammation of the cerebrum. The consideration that guanadin normally produced in the body was dealt with and rendered harmless by the liver,
led Fuchs to try the effect of ‘side-tracking’ the liver by means of an Eck’s fistula, as Pawlow had done years before for quite a different investigation. The effects were almost identical with those of guanidin feeding, but more intense, though no poison was introduced from without, the intoxication being purely endogenous.

The idea that changes in the nervous system might be produced by endogenous toxins was of course not new, but precise evidence was hard to obtain and to interpret. Such evidence as there was cast suspicion on the liver. In 1912 Wilson, in his study of progressive lenticular degeneration, had considered it probable that the changes in the brain in that condition were due to the selective action of a toxin formed in the liver or associated with hepatic disease; pseudosclerosis, too, was known to be associated in some cases with a hepatic degeneration. The work of Fuchs showed that liver insufficiency was capable, by itself, of producing nervous disease—almost certainly by endogenous toxins.

The brains and cords of the animals employed have been carefully investigated by Pollak, and the study of them forms the basis of his article. They show a general inflammation, exactly the same whether produced by guanidin feeding or by insertion of the Eck’s fistula, and in no way differing from that found in encephalitis lethargica. Pollak describes these changes in detail. He first shows that they constitute inflammation, in the strictest sense of the word, all the essential pathological factors being present; he then describes separate details, laying emphasis on the perivascular infiltration with mononuclear cells, the dropping out of ganglion cells, and neuronophagia; he believes that while many of the cells found clustered round damaged ganglion cells are true glia cells, many also are blood elements derived from fine capillaries; in the subeortical ganglia he finds degenerative changes and distinct evidence of hemorrhages; in the cerebellum there is no change, but the cord shows in a less degree the same changes as are present in the cerebrum.

This study constitutes a step towards the understanding of the relation of the liver to the central nervous system, but the encephalitis produced does not seem to be in any way characteristic.

J. P. MARTIN.


As regards the cerebrospinal fluid, observations were made on five cases and the previous results of De Lavergne were confirmed, viz., the fluid invariably shows change in albumin, cells, and sugar content. On the other hand, investigation into the question whether the fluid contained either toxin or antitoxin gave completely negative results. As regards the blood, antitoxin was invariably present, and the investigations show that this represents an active immunizing process, irrespective of whether serum had or had not been administered.

W. JOHNSON.
The changes in cerebrospinal fluid in diphtheritic paralysis


According to certain French authors the toxins of the diphtheritic bacillus travel via the nervous elements of the peripheral nerves. For certain English authors (Orr and Rows, Walshe) the path is rather the lymphatic channels surrounding the peripheral nerves. From the numerous examinations of the cerebrospinal fluid in cases of diphtheritic paralysis, the following facts emerge. The fluid is clear, and to the naked eye normal in appearance. Its pressure is rarely increased. The Klebs-Löffler bacillus has not been demonstrated. Slight lymphoeytosis is not infrequently found (occasionally up to 15 or 30 per c.mm.), but never polymorphonuclear cells. The number of lymphocytes bears no relation to the severity of the clinical paralysis. The albuminous content is always increased to a greater extent than might be expected from the lymphocyte count. This dissociation between albumin content and cells appears to be a characteristic of the meningeal reaction. The sugar content in the fluid is actually increased.

The author attributes these changes to the circulation of the toxin in the blood and to the irritation of the fluid secretory apparatus—chiefly the choroid plexuses—whereby abnormal cerebrospinal fluid is formed. No observations are so far available to show whether in cases of diphtheria, without paralysis, similar alterations in the fluid occur.

W. Johnson.


Diphtheritic paralysis is too generally attributed to a peripheral neuritis. Philippe and Barbonneix have shown that the nerve centres suffer as well as the peripheral nerves. Aubertin supports this view, and points out that, in addition to the usual picture of peripheral nerve palsies, it is not infrequent to find the occurrence of partial muscle group palsies and atrophies which definitely indicate an affection of anterior horn cells. The cerebrospinal fluid, too, shows alterations due to meningeal reaction (Chauffard and Lecomte).

The two patients shown presented paralysis of accommodation, phonation, and deglutition, together with weakness of all four extremities. In the upper limbs, all muscles—flexors and extensors together with the small muscles of the hand—were affected. In the lower limbs the extensor groups were less affected than the flexor, and this was also true of the trunk muscles. The authors contrast this with the usual picture of peripheral neuritis, and term it 'the spinal form of diphtheritic paralysis'. The sensory loss in these two patients is described as of segmentary distribution. The authors regard the occurrence of this poliomyelitis as indicating a severe intoxication, while polyneuritis indicates a milder condition. In
using the term poliomyelitis, they are careful to point out that the pathological process is much less severe than in anterior poliomyelitis, and that complete recovery may be expected.

Incidentally they note the comparative frequency of diphtheritic paralysis amongst adults in recent years; but whether this is due to the more universal use of serum or to an alteration in the type of virus it is difficult to determine.

W. Johnson.

SYMPATOMATOLOGY.


Numerous authors, especially of the French school, have reported an increase in the percentage of glucose in the cerebrospinal fluid in cases of encephalitis lethargica, as well as in other conditions affecting the mesencephalon. In a number of cases of the former disease the amount of glucose varied from 70 to 100 mgm. per 100 c.c. of cerebrospinal fluid. This was contrasted with Mestrezat's figure of 53 mgm.—the average of 11 cases where the cerebrospinal fluid was removed at operation prior to stovaine anaesthesia. Other observers have found the normal to be higher: von Jaksch 60 to 80 mgm., Kraus and Corneille 80 mgm., A. H. Hopkins 60 to 75 mgm., Weston 60 to 70 mgm. The variation in these results is probably due to difference in technique.

Coope examined 95 cerebrospinal fluids for glucose by the method of Folin and Wu. No normal fluids were examined. The glucose percentage in the cerebrospinal fluid of 11 cases of lethargic encephalitis varied from 54 to 94 mgm., with an average of 74 mgm. In 51 cases of mental disease the figures varied from 44 to 102 mgm., and in a case of imbecility a reading as high as 111 mgm. was obtained. On the other hand, in 12 cases of tuberculous meningitis the glucose in the cerebrospinal fluid varied from 14 to 53 mgm., with an average of 28 mgm., a reading above 40 mgm. being obtained in only one case. An examination of the glucose content of the cerebrospinal fluid is therefore of value in distinguishing encephalitis lethargica from tuberculous meningitis, but it is doubtful if it gives as much information as the simpler examination of the chlorides in the cerebrospinal fluid. It would have been interesting to know to what extent the glucose in the cerebrospinal fluid varied with that of the blood, but unfortunately the latter was not examined.

J. C. Greenfield.


A brief report of a case of hemiplegia in a boy, age 12. The onset was apoplectiform in the course of a septicæmia which terminated favourably.