Nine of these patients were sporadic but consecutive cases of clinically atypical pneumonia, of whom seven had an E.S.R. of over 90 mm. on admission and four had rates of 120 mm. or more at some stage of their illness. These levels are much higher than those commonly seen in acute pneumonia.

The high sedimentation rates are not due to the excessive aggregation of red cells produced by the cold agglutinins. The plasma protein abnormalities which occur do not appear to be unusual or specific to this form of pneumonia.

Such very high rates can be demonstrated only by the Westergren method; the Wintrobe method may give disproportionately low or even normal results.

If a patient with acute uncomplicated pneumonia has an E.S.R. of over 90 mm. the diagnosis of primary atypical pneumonia should be considered, especially if there are any other unusual features.

We are most grateful to Dr. Neville Oswald for his advice and to the physicians of St. Bartholomew's, the Royal Free, the Royal South Hants, and St. Thomas's Hospitals for their permission to include details of patients who had been under their care.

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PRIMARY ARACHNOID CYSTS

REPORT OF TWO CASES

BY

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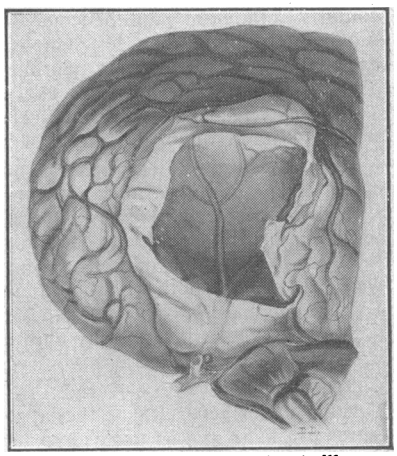
Richard Bright (1831) gave a short account of cysts arising in relation to the surface of the brain, and there is little doubt that some of them were cysts of the arachnoid, as the accompanying reproduction of his original illustration shows. A long time elapsed before any further noteworthy contributions to this subject were made.

de Martel and Guillaume (1930) reported seven cases of arachnoid cysts in the posterior fossa and stressed the sudden onset of headache with neck stiffness, suggesting the diagnosis of meningitis or subarachnoid haemorrhage. Mullin (1932) described two cases in which a cyst in the cerebellopontine angle produced a syndrome resembling that of an acoustic neurinoma: arachnoid cysts are commonly found associated with acoustic tumours, but in Mullin's cases the internal auditory meatus was clearly exposed and a tumour excluded. The membrane was not examined microscopically; therefore it is possible that the cysts were ependymal in origin as in a case described by Hardman and Jefferson (1938).

Characteristics

Tönnis, at the meeting of the Society of British Neurological Surgeons in Berlin in June, 1937 (unpublished), described his experience of eight cases of "congenital cysts of the cisterns" simulating intracranial tumours. This was a most valuable contribution in which the characteristics of arachnoid cysts were defined as follows: The membranes correspond to the meninges in their structure; signs of inflammation in the cyst wall are absent; the cystic fluid is clear and usually xanthochromic, and coagulates spontaneously after removal.

McConnell and Douglas (1939), writing on intracranial cysts, included examples of what they termed "subarachnoid cysts." Their illustrations make it clear that the outer wall of the cysts was composed of arachnoid, and the inner wall of pia mater. Three examples were cited: in the first the cyst occupied most of the right anterior fossa, in the second the anterior half of the right temporal fossa, and in the third the midline of the posterior fossa. In all three instances it appeared as though the underlying brain was deficient. Macroscopically the arachnoid and pia forming the cyst walls were normal. The fluid of the cysts was not analysed and there was no microscopical examination of the membranes.



Reproduction of Richard Bright's illustration of an arachnoid cyst in the left Sylvian fissure. (Kindly lent by Mr. James Hardman.)

Horrax (1924) reported some cases of Cushing's of midline cysts of the posterior fossa causing increased intracranial pressure. The cysts, however, appeared to be dilatations of the cisterna magna, and they were attributed to inflammatory adhesion of the membranes, though evidence of inflammation was lacking in most of the cases; examples of cysts in the cerebellopontine angle associated with otitis media were also encountered. Cases similar to Cushing's were described by Allen and Corkill (1937); in two of them the cysts were apparently separate from the cisterna magna and were therefore true arachnoid cysts. These authors also favoured the inflammation theory, though no histological or other evidence was given to support it.

Successful removal of a circumscribed cyst of the leptomeninges involving the spinal cord and cauda equina was reported by Spiller, Musser, and Martin (1903). The walls of the cyst were described as transparent, but again no histological examination was carried out; in a subsequent paper, however, Spiller (1909) attributed such lesions to inflammation, and hence the origin of the term "circumscribed serous spinal meningitis." Spiller was supported in his belief by Horsley (1909), who reported his cases under the title of "chronic spinal meningitis"; he suggested that in some instances syphilis or gonorrhoea was the responsible infection.

Stookey (1927) believed there was no justification for the view that arachnoid cysts are of inflammatory origin, though he reported his own case under the title of "Adhesive Spinal Arachnoiditis."

Scrutiny of case records in the literature lends abundant support to Stookey's opinion, but histological reports on the membranes are rarely given. Therefore it seems worth

while to record the following cases in which detailed description of the cysts and histological reports are available.

Case 1

A man aged 21 was admitted to hospital on May 16, 1941, complaining of headache, vomiting, and diplopia. Ten weeks previously he had received a severe blow on the chest while playing football. Two weeks later he began to have headaches, mostly frontal, but sometimes occipital; the frontal headaches extended behind the eyes. He felt sick in the morning and sometimes vomited. For four weeks diplopia had occurred on looking to the right. There was nothing relevant in the past and family histories.

On examination the following abnormal signs were found: bilateral papilloedema (one dioptre); dilated pupils, right more than left; slight weakness of the right external rectus; left upper neurone facial weakness; and absence of abdominal reflexes except the right lower one. The plantar responses were unaffected. Nothing abnormal was discovered on examination of the other systems.

Investigations.—Radiography of the skull showed decalcification of the posterior clinoid processes. Lumbar puncture done elsewhere had revealed a pressure of 300 mm.; tests for protein, chlorides, and sugar were normal, and there was no excess of cells. The Wassermann reaction was negative in both the blood and cerebrospinal fluid. Electroencephalography suggested a lesion in the *left* parietal lobe, but ventriculography showed displacement of the ventricles to the *left*.

Operation (May 17).—A right fronto-temporal flap was elevated under general anaesthesia. As the dura mater was extremely tense it was decided to tap the anterior horn of the right ventricle, but a small incision in the dura revealed a bluish-looking membrane beneath it. A subdural haematoma was suspected, but a brain cannula passed through the membrane showed yellow fluid. A sample was sent for examination. A further quantity of fluid was withdrawn to slacken the dura, which was then opened. The Sylvian fissure was widely separated by a large cyst extending well forward into the anterior fossa and downward into the middle fossa. When the cyst was opened the island of Reil was clearly visible, with many tortuous branches of the middle cerebral artery running across its surface. The outer or superficial wall of the cyst was formed by a *uniformly transparent* membrane which appeared to be continuous with the arachnoid around the periphery of the cavity, where it was fused with the inner wall formed by macroscopically normal pia mater and underlying brain. A small entirely separate loculus at the anterior end of the middle fossa was separated from the rest of the cyst by a very thin membrane. There were no adhesions between the cyst and the dura mater. The superficial wall of the cyst was removed and preserved for histological examination; the deep wall, being composed of pia mater, was left undisturbed. There was no tendency at all for the brain to expand and fill the space left by the removal of the cyst; the appearance was as though part of the cerebrum was missing.

The patient made a rapid recovery. By July 14 he was free from symptoms, and the papilloedema and other physical signs had disappeared, except that the left abdominal reflexes were not so brisk as those on the right. Air encephalography, performed through the cisternal route eight weeks after operation, showed the ventricles to be normal in shape and position, but they were about twice the size observed before removal of the cyst. A large amount of air had collected in the space previously occupied by the cyst.

Examination of cyst fluid on May 17 showed slightly blood-stained amber fluid with a total protein of 460 mg. per 100 ml. (considerable excess of globulin, Pandy plus 4, Nonne–Apelt plus 3). There were 13 white cells and 22,664 red cells per c.mm.

Histology of Cyst Wall (Professor Dorothy Russell).—"The wall of the cyst is composed of arachnoid membrane thickened by fibrosis, and showing (*a*) considerable recent focal haemorrhage, and (*b*) areas of greatly increased vascularity associated with chronic inflammatory infiltration and considerable numbers of macrophages containing iron pigment. A piece of dura mater overlying the cyst showed no abnormality. The appearances are consistent with a traumatic origin, but traumatic changes in a previously existing arachnoid cyst cannot be excluded."

Case 2

A boy aged 10½ months was admitted to hospital on May 16, 1947, because of enlargement of the head and vomiting. Labour had been difficult, lasting 51 hours. The head was observed to be larger than normal at birth, but was not measured. There had been four bouts of vomiting over the period of five months before admission. There was no history of meningitis or head injury.

On examination the maximum circumference of the head was found to be 23¼ in. (59.6 cm.) (normal for the age, 17–18 in. (43–45.5 cm.)), the fontanelles were large and tense, and the cranial sutures were widely separated. The limited neurological examination possible at this age was negative.

On May 20 ventriculography was performed with needles passed through the lateral angles of the anterior fontanelle. Only 40 ml. of C.S.F. was replaced with air; the lateral ventricles were shown to be very dilated but not displaced. Though the third ventricle was not visualized it was decided to proceed immediately with exploration of the posterior fossa. 4 ml. of 0.4% indigo carmine was injected into the ventricles so that the patency or otherwise of the aqueduct could be determined at operation.

Operation (May 20).—Under infiltration anaesthesia, a midline muscle-separating posterior fossa exposure was carried out. When the bone was removed a bluish-looking membrane could be seen through the thin dura mater on the right side. A small incision was made in the dura and the cyst tapped. Clear colourless fluid was withdrawn; *there was no trace of indigo carmine in this fluid.* The dura mater was then reflected and the cyst was opened; there were no adhesions between the dura and the cyst. Unfortunately the fluid escaped too quickly for any to be sent for analysis. Pressure on the anterior fontanelle did not cause any further fluid to flow from the cyst, but instead it caused ballooning of the cisterna magna, which, by aspiration through a hypodermic needle, was shown to contain dye. *Thus the cyst was proved to be separate from the cisterna magna.* The outer wall of the cyst appeared to be fused around its periphery with the pia mater covering the cerebellum; and the latter was displaced upward and to the left.

When seen four years after the operation the patient was speaking normally but was only just beginning to walk unaided. The maximum circumference of his head was then 23 in. (58.4 cm.), a fraction of an inch less than at the time of operation.

Histology (Dr. E. A. Atkinson).—The tissue received had the appearance of normal arachnoid membrane; there was no evidence of inflammation.

Discussion

These two records demonstrate clearly the strict criteria of primary arachnoid cysts; the outer walls were formed by arachnoid membrane, as confirmed by histological examination, and the inner walls by the pia mater of the underlying brain surface. In Case 1 it was possible to collect the fluid; it was xanthochromic and contained a level of protein well above that of the cerebrospinal fluid; in Case 2 there was also proof of the nature of the membranes, and though the fluid was not analysed it was conclusively demonstrated by the dye technique, and by pressure on the

anterior fontanelle, that the cyst was not a distended cisterna magna and that it did not communicate with the subarachnoid space beyond its boundaries.

There was no evidence in the histories, at operation, or on histological examination, of a bacterial meningitis or neoplastic process. There was no history of direct head injury in either instance, but in Case 1 there had been a blow on the chest a few weeks before the onset of symptoms. It is well recognized that compression injuries of the thorax sometimes cause secondary damage to the brain (Rowbotham, 1945). It appears likely that the changes in the arachnoid reported by Professor Dorothy Russell in this instance were of traumatic origin, as she suggests, and that the symptoms were thus precipitated by the injury; but it is inconceivable that the large cyst, insinuating its way deeply in the Sylvian fissure, could have arisen solely from the effects of a recent injury; in support of this assertion is the fact that air encephalography carried out eight weeks after operation showed persistence of the space formerly occupied by the cyst—an unlikely finding if the brain had been merely displaced by a traumatic cyst.

Conclusion

The cause of the majority of arachnoid cysts is unknown; the term "primary arachnoid cysts" is suggested for this group and "secondary arachnoid cysts" for those which can be attributed to injury or inflammation, and for those which are associated with tumours.

It may be that primary cysts result from an error of development of the leptomeninges and the underlying nervous tissue.

In the cases reported by McConnell and Douglas (1939) and in my first case the cysts appeared to occupy an irregular space, giving the impression of absence rather than displacement of nervous tissue, and there was no tendency for the cavity to disappear after removal of the cyst; in Case 1 the space was demonstrated by air encephalography eight weeks after operation—unlikely if the brain had been simply displaced. It seems impossible to escape the conclusion that the cyst in this case was congenital.

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SYMPTOMS AND CLINICAL ASPECTS OF DIABETES MELLITUS

BY

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The division of cases of diabetes mellitus into relatively well-defined groups has simplified the clinical picture and led to greater success in the management of the disease. The response to insulin was the basis of Himsworth's (1939) classification. In general, the young and thin patient tended to be insulin-sensitive; the elderly and obese patients with hypertension tended to be insulin-resistant. Further, the onset of the disease in the sensitive diabetic was generally sudden and severe, and ketosis rapidly developed if insulin was withheld. In the insensitive group the onset was usually insidious and ketosis was rarely present.

Plasma insulin estimations confirmed clinical observations that all diabetic children, most adults under 40, and many over 70 are insulin-deficient (Bornstein and Lawrence, 1951a, and 1951b). In the insulin-resistant group plasma insulin levels are near normal, and Lawrence (1951, 1954) employed the terms "lipoatrophic" and "lipoplethoric" diabetes to describe the two opposite examples encountered. Lipoatrophic diabetes is so rare that it can well be regarded as "a collector's piece." In contrast, the lipoplethoric type accounts for the majority of obese middle-aged diabetics, being twice as common in women as in men.

The essential purpose of the present investigation is to determine the incidence of symptoms in the ketosed and non-ketosed groups of diabetic patients. A study is also made of the way each case presented, the clinical picture, and the response to treatment.

Method and Material

The paper is based on the case histories of 300 patients attending the diabetic clinic at the Royal Devon and Exeter

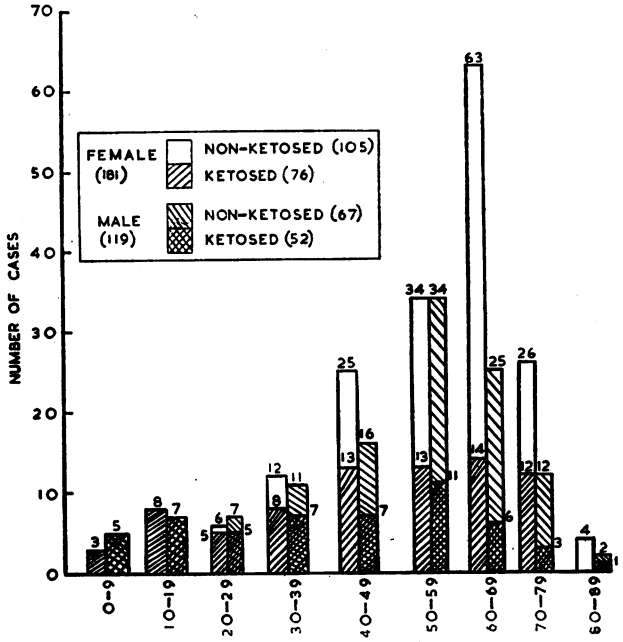

FIG. 1.—Age distribution of 300 diabetics.