SEROUS MENINGITIS*

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NEUROLOGISTS and neurosurgeons have long been troubled by a heterogeneous group of patients who showed evidence of increased intracranial pressure but who proved not to have space-occupying lesions. Some of these patients have a history of an infection contiguous to the intracranial cavity in ears, mastoids, or sinuses. They are suspected of having a brain abscess. A few have had an injury to the head and are thought to have subdural hematomas. Others have no history of infection or injury and are considered to have brain tumors.

These cases have been variously referred to as “pseudo-tumors,” serous arachnoiditis, serous meningitis, chronic arachnoiditis, toxic hydrocephalus, otitic hydrocephalus, meningitis sympathica, aseptic meningitis and so on. The wide variety of terminology clearly indicates the general confusion regarding them.

Quincke reported ten cases of serous meningitis and emphasized the spinal fluid findings and the increase of intracranial pressure. Passot, in a thesis, distinguished between the purulent and non-purulent complications of otitis media. He differentiated them by spinal fluid examination. The non-purulent group was considered due to an excess of cerebrospinal fluid in the subarachnoid spaces. Warrington in 1914 described five groups of intracranial serous effusions of inflammatory origin: those due to otitis media, to tuberculosis, to specific fever, to head injury, and finally an unexplained group. As his title indicates, he considered a near or distant focus of infection as the primary origin. Claude discussed the subject thoroughly. He gave trauma, infection, and encephalitis as the etiologic factors and emphasized the value of air studies in differential diagnosis.

More recently Symonds, Davidoff and Dyke, McAlpine, Howell, and Sahs and Hyndman have contributed to this subject. The majority of authors have advocated conservative treatment, once the condition was recognized, with repeated spinal drainages and dehydration of the patient. Davidoff and Dyke, in contrast, advocated subtemporal decompression as a routine measure. The need for air studies to differentiate space-expanding lesions is admitted by most authors, although Symonds did not use them in his three cases and McAlpine in only two of his five cases.

It is obvious from a review of the literature that these patients all recovered spontaneously or at least with conservative treatment. However, in many instances it is not clear as to whether impairment of vision occurred in the patients who were treated expectantly or conservatively. McAlpine

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REPORTS MANINGITIS

reports optic atrophy in one of his cases and it is to be suspected that this complication may have occurred more often than had been reported.

CASE REPORTS

Case 1. An early experience with a case of "pseudo-tumor" was that of Mrs. J. H. G., a 49-year-old woman, first seen in February 1935. At that time she complained of failing vision, nausea and vomiting, and dizziness during the preceeding year with tinnitus for one month. The past history was not relevant except for periodic frontal headaches occurring on an average of once monthly since the age of 18. There had been no change in the intensity and frequency of these headaches during the preceeding year. General physical and neurological examinations revealed no significant findings except bilateral papilledema of 3 to 4 diopters with reduction of vision to 0.2 in each eye.

Under the assumption that we were dealing with an unlocalized brain tumor, ventriculography was performed. Both ventricles were tapped and each contained about 15 cc. of fluid. X-rays taken after replacement of the fluid by air failed to disclose any abnormalities of the ventricular system. A few days later formal right subtemporal decompression was carried out. The brain appeared normal but the leptomeninges were thought to be somewhat thickened and opaque, and there was an unusual amount of subarachnoid fluid.

Convalescence was uneventful and the patient left the hospital on the 17th postoperative day. She lives in another city and has not been seen since three months after the operation. At that time the margins of the optic discs were blurred but there was no elevation. Vision was 1.0 in the right eye and 0.8 in the left. The decompression bulged moderately. We have heard from her at intervals since; the last report was in August 1944. Her vision had not changed. There was still slight bulging of the decompression, indicating the persistence of a mild degree of increased intracranial pressure.

Comment. It does not seem likely that this woman's vision would have been spared without decompression. No etiologic factors for her intracranial condition were ever discovered.

Case 2. A similar ease, but with chronic infection present elsewhere in the body as a possible etiologic factor, was that of F. H., an 11-year-old boy seen on the children's orthopedic service at the Cook County Hospital in March 1935. He had been under treatment for chronic osteomyelitis of the left femur and humerus of 2 years' duration. Headaches had been present for 3 weeks, and 2 weeks before he had developed diplopia and begun to vomit frequently. Examination revealed slight weakness of the right side of the face, bilateral abducens palsy, and bilateral papilledema of 2 to 3 diopters. X-rays of the chest were negative.

He was thought to have a metastatic brain abscess, and ventriculography was performed. The ventricles appeared to communicate at the time of the air injection but the roentgenograms showed poor filling of the left ventricle with no air in the third. A left subtemporal exploration and decompression were carried out. There was more than the usual amount of subarachnoid fluid but the brain itself appeared normal without any evidence of an intracranial lesion. When a cannula was introduced into the temporal lobe, the temporal horn of the ventricle was readily entered and about 80 cc. of clear fluid were aspirated.

The child made an uneventful convalescence and the papilledema and abducens palsy rapidly receded. Bulging of the decompression was marked for the first month; the bulging could be collapsed temporarily by spinal drainage. Gradually the decompression ceased to bulge and by dismissal 2 months after operation it was flat.

Comment. This patient might well have been treated by conservative means. However, the ventriculograms misled us as they appeared to substantiate the clinical impression of a left temporal lobe abscess. Accordingly surgery was performed without any further delay.
Case 3. A very striking case was that of Mrs. G. R., a 44-year-old white woman, first seen in October 1937. She had been admitted to the hospital one month before because of headaches and pain over the left mastoid of 2 months’ duration. Bilateral otitis media had been present since childhood and a right mastoidectomy had been performed a year before. The patient had recurrent chills, with her temperature rising to 103 at times. She complained of severe headache and vomited frequently. After treatment with sulfonamides for a few days her condition improved and a left mastoidectomy was performed. Two days later the chills recurred and her fever rose to 104. On the 4th day after the mastoidectomy the left lateral sinus was explored. Free bleeding occurred from the upper end but not the lower, so that the internal jugular vein was ligated in the neck. Following this procedure the temperature fell to normal and the patient’s general condition improved. However, drowsiness and at times stupor appeared; she complained again of severe headache and vomited frequently. Three weeks after the operation on the lateral sinus the patient was found to have bilateral papilledema of 2 to 3 diopters, and neurosurgical consultation was called.

Neurological examination revealed slight weakness and incoordination of the right extremities and slight weakness of the left lateral rectus muscle. The spinal fluid pressure was 320 mm.; the fluid was clear, and contained only 3 cells. The case was not thought to be one of abscess and treatment was conservative for 2 weeks. During that time several spinal punctures were made with removal of 20–30 cc. at each tap. The pressures ranged from 150–500 mm. of water. At the end of that time the papilledema was observed to be increasing with several fresh retinal hemorrhages. There was no progression in the neurological findings and perimetric fields were normal for both form and color.

Ventriculography was attempted. Both lateral ventricles were tapped, but only a few cc. of fluid were obtained from each and air injected escaped immediately from the cannulae; X-rays failed to show any air in the intracranial cavity. Following this the patient had severe headache, was restless, and claimed that she could not see. She was given 250 cc. of 25 per cent sucrose intravenously. The next day her general condition was good. The pupils reacted to light but the patient denied light perception. The spinal fluid pressure was 450 mm. of water.

Left subtemporal exploration and decompression were carried out the following day. The brain was under marked tension when the dura was opened but there was no widening of the convolutions. The brain was injected and there was a grey-yellow exudate along the larger cortical vessels. Exploration of the temporal lobe with a cannula yielded no evidence of an abscess.

Convalescence was uneventful from a general standpoint. The patient was confused and irrational for several days. Spinal punctures continued to show fluid under pressure of 300–400 mm. Cultures were repeatedly negative. At dismissal on the 12th postoperative day the patient was normal mentally, the decompression bulged moderately, the fundi showed 2–3 diopters of elevation bilaterally but all hemorrhages and exudates were absorbed, and the patient could read newspaper print.

Her later course has been of the greatest interest. The papilledema slowly receded and disappeared within 6 weeks after dismissal. At no time has it recurred. Vision was 0.8 in each eye 6 weeks after dismissal and has remained at that level. Moderate to marked bulging of the decompression was present during the first year and the patient complained of persistent headaches. Drainage persisted from both ears. At the end of that time a radical mastoidectomy was done on the right side. Since then she has had very little drainage from the right ear and much less headache and bulging of the decompression.

Two years after her decompression she had an uneventful pregnancy and delivered a normal child. The pregnancy had no effect on the bulging of the decompression. Three years later she had another child. Gradually the decompression has ceased to bulge significantly, although it is never completely flat. She occasionally has a recurrence of drainage from both ears, often as a result of an upper respiratory infection. At such times headaches recur and the decompression bulges markedly. When last seen in December 1944, over 7 years after her de-
SEROUS MENINGITIS

compression, there was still very slight bulging of the decompression. She had no complaints, vision was 0.8 in each eye and the fundi were normal.

Comment. The prolonged increase of intracranial pressure and the clear relationship of its exacerbations to the exacerbations of the chronic otitis is very striking. It is very doubtful if this patient’s vision could have been preserved without decompression.

Case 4. Another case of clear otitic origin was that of R. C., an 8-year-old girl seen on the otologic service at the Cook County Hospital in April 1938. Three months before she had had right mastoiditis and lateral sinus thrombosis for which mastoidectomy and ligation of the internal jugular vein had been performed. Bilateral papilledema had been noted since that time.

Neurological examination was negative except for bilateral papilledema of 2 to 3 diopters and enlarged blind spots in the perimetric fields. Vision was 1.0 in each eye.

Ventriculography was performed and communication of the lateral ventricles was demonstrated. The roentgenograms showed poor filling of the right lateral ventricle, and a right subtemporal exploration and decompression were performed. The cortex of the temporal lobe was injected and the brain was under increased pressure. However, exploration of the temporal lobe with the brain cannula showed no evidence of abscess.

The child's convalescence was complicated by epidemic parotitis but she left the hospital on the 23rd postoperative day. She was seen at intervals after this and within 3 months the fundi were normal. The decompression continued to bulge for 3 or 4 years. When the child was last seen in 1948, over 5 years after operation, she was perfectly well, the operative site was depressed and she requested repair of the skull defect. After the lack of necessity for that was explained to her she was satisfied.

Comment. Localization of this type of lesion to the posterior fossa produces an internal hydrocephalus with a clinical and ventriculographic picture that suggests posterior fossa tumor. Horrax differentiated between chronic serous arachnoiditis of a generalized character and chronic circumscribed or cystic arachnoiditis, a localized process which he considered the sequel of a meningoencephalitis. He reported 33 cases of generalized chronic serous arachnoiditis of the posterior cistern which he divided into acute or subacute, chronic and otitic cases.

Case 5. The first experience with arachnoiditis of the posterior fossa was the case of E. F., a 14-year-old girl first seen in November 1935 at the Cook County Hospital. Two years before she had complained of stiff neck and headache and, under a diagnosis of cervical Pott's disease, was kept in bed for 8 months. She was then well until a month before, when there was sudden onset of headache and great irritability and restlessness. She had improved after two spinal drainages.

Examination showed a frail blonde child who appeared to be only about 10 years of age with no development of secondary sex characteristics. She was unable to stand or walk without support and there was marked hypotonicity of all the skeletal muscles. There was a bilateral Hoffmann sign and the Chaddock, Gordon and Oppenheim signs were present bilaterally. No Babinski could be obtained but there were a few jerks of ankle clonus on the left. There was bilateral pallor of the optic disc with moderate loss of substance. Vision was reduced to 0.01 in the right eye and 0.1 in the left. The child was talkative, euphoric and facetious with frequent changes of mood. She was apparently somewhat precocious but had little insight.

The spinal fluid was slightly xanthochromic and under marked pressure. It contained no cells, and a guinea pig inoculated with it failed to develop tuberculosis. X-rays of the skull
showed marked pressure digitations, separation of the sutures, and erosion of the posterior clinoid processes.

She was thought to have a brain tumor but the localization was not clear. Ventriculography demonstrated a marked symmetrical internal hydrocephalus. This was thought to indicate a posterior fossa tumor and a suboccipital craniectomy was carried out. There was no evidence of tumor in the posterior fossa but the arachnoid over the posterior cistern was thickened and opaque. When it was opened the fourth ventricle was seen to be considerably dilated. The arachnoid of the posterior cistern was widely resected and the wound was then closed.

Convalescence was satisfactory. At dismissal she could walk but was moderately ataxic. The mental attitude was the same and the visual acuity had not improved.

She was seen 2 years later and presented at a clinic. The decompression was flat and there was no ataxia. Visual acuity was 0.1 in the right eye and 0.6 in the left. She seemed perfectly well at this time. However, a month later she returned to the hospital complaining of headache, blurred vision and stiff neck. The decompression bulged markedly but could be collapsed by spinal drainage, which gave the patient marked relief. The spinal fluid contained 8 lymphocytes.

An attempted encephalogram was unsuccessful. A ventriculogram showed marked internal hydrocephalus. The fourth ventricle was visualized and markedly distended. Re-exploration was carried out. Exposure of the posterior cistern and fourth ventricle was very difficult because of adhesions. The posterior cistern was obliterated except for several pockets of clear fluid. The child did not rally after operation and died the following day.

Comment. Horrax\(^5\) reported a death in his series after operation for recurrence of symptoms.

Case 6. Another patient with posterior fossa arachnoiditis is still under observation with symptoms that suggest the process is present in other parts of the intracranial cavity. R. H., a 40-year-old woman, was first seen at Mercy Hospital in April 1941. She had had multiple operations in the past including an appendectomy, thyroidectomy, and cholecystectomy and had been under treatment for peptic ulcer and colitis for the preceding 3 years. A thrombophlebitis of the left lower extremity had followed the cholecystectomy.

Recurrent headache with associated vomiting had been present for 2 years. For the preceding 2 months there had been more severe and pronounced headache, and dizziness, even in the horizontal posture, had developed. Blurring of vision had been present for 1 month.

Neurologic examination was essentially negative except for slight increase of the tendon reflexes on the left with a few jerks of ankle clonus on that side. There was bilateral papilledema of 2 to 3 diopters which the ophthalmologist felt had been present for some time. Vision was 0.8 in each eye and the perimetric fields were normal.

A ventriculogram revealed a symmetrical internal hydrocephalus. The patient was thought to have a posterior fossa tumor and a suboccipital craniectomy was performed. When the dura was opened there was a large arachnoid cyst situated in the midline over the posterior cistern and separating the lower part of the cerebellar hemispheres. The cyst measured approximately 3 X 3 X 5 cm. and contained clear fluid. After its evacuation, thorough exploration of the posterior fossa failed to reveal any neoplasm and the wound was closed. The dura, perhaps unwisely, was sutured.

Microscopic examination of the wall of the cyst revealed moderate thickening of the arachnoid with occasional small plaques of cellular proliferation and a rare cystic space. The pathologic diagnosis was chronic, non-specific arachnoiditis with inclusion cysts.

The patient's convalescence was prolonged. Headaches, vomiting, and dizziness persisted. Spinal drainages on numerous occasions revealed manometric pressures ranging from 190 to 400 mm. of water. The total protein was 100-120 mg. and there was a persistent pleocytosis of 140-416 cells which were all lymphocytes. Repeated cultures failed to show any bacterial growth. She was finally dismissed on the 40th postoperative day.
She has been seen at frequent intervals. There has never been any bulging of the suboccipital decompression. The papilledema slowly receded but slight elevation of the discs has persisted and is still present. The spinal fluid pleocytosis finally disappeared. After 8 months the patient returned to her work as a secretary, but frequent headaches and vomiting have persisted. These are temporarily relieved by spinal drainages or intravenous injections of hypertonic sucrose solution. The spinal manometric pressure is always increased and is usually 300–400 mm. of water. An encephalogram in June 1943 revealed the symmetrical internal hydrocephalus to be present still, with the ventricles a little larger than 2 years before.

Her vision remained at 0.8 in both eyes until recently, and no defects of the visual fields were noted. However, her ophthalmologist has recently found her vision to be reduced to 0.5 O.U. and the perimetric fields now show a partial upper quadrant homonymous defect.

Comment. It is now felt that this patient’s arachnoid cyst with consequent obstructive hydrocephalus was only part of the process of generalized chronic serous arachnoiditis. It would appear that she may suffer from another phase of that condition, opticocochiasmal arachnoiditis, which Lillie\(^7\) has described. In retrospect it was a mistake to close the dura at the time of the operation. If the dura had been left open, persistent bulging of the suboccipital decompression would probably have occurred and the patient’s course might have been much better. A transfrontal exploration of the optic chiasm with generous subtemporal decompression seems indicated now that vision has begun to fail and field defects have appeared. However, we have been reluctant to undertake further surgery on this woman.

DISCUSSION

These six cases illustrate some of the problems in a group of patients who can be briefly described as having subjective symptoms suggesting increased intracranial pressure and showing papilledema. Neurological findings, exclusive of those associated with increased intracranial pressure, are minimal and definite localization is seldom suggested. A near or distant focus of infection may be present. If so, abscess is correspondingly suspected; if not, neoplasm is the favored diagnosis. The cerebrospinal fluid manometric pressure is high; the fluid is clear or contains only a few lymphocytes. Air studies fail to show any evidence of a space-occupying lesion unless the arachnoiditis involves the posterior cistern; in that case an internal hydrocephalus is demonstrated. In this last group the indication for operation is clear; unfortunately the results are not always satisfactory. Horrax\(^5\) reported five deaths in 33 cases within two years after operation. Of the remaining 28 he classified 14 as improved and 14 as well. Barre\(^1\) reported three cases of arachnoiditis of the posterior fossa that were operated on with one death.

Examples due to trauma have not been included in this paper. However, on the neurosurgical service at the Cook County Hospital patients are frequently seen who develop choked discs after head injury. Other signs of subdural hematoma are absent. Some of these patients have been explored with negative results; others have been treated conservatively with dehydration and spinal drainages and the papilledema has subsided and the subjective symptoms disappeared. It is difficult to say whether the pathology in these
cases is an inflammatory reaction in the leptomeninges secondary to trauma, or a late chronic cerebral edema, or whether there is some other explanation. It is my impression that these patients usually have suffered subarachnoid hemorrhage, but, on the other hand, many cases of traumatic or spontaneous subarachnoid hemorrhage fail to show any evidence of papilledema.

This whole subject is very troublesome and difficult for the neurosurgeon. The cases that I have presented represent only one group of the heterogeneous "pseudo-tumors," that with objective evidence of increased intracranial pressure. An even more obscure and difficult type is the patient who has headache and often other subjective evidence of intracranial pathology, with perhaps some objective neurological findings, but who does not have objective evidence of increased intracranial pressure. Some of these patients have convulsive seizures which may be quite localized in nature. Air studies may be misleading, as in Cases 2 and 4. Exploration reveals no evidence of neoplasm or perhaps no gross pathology at all. In some cases there are localized thickening and opacity of the arachnoid, localized shrunken or atrophic areas of cortex or combinations of both.

Another type is the localized arachnoiditis of the optic chiasm. Here, as a rule, the symptoms and objective findings point to a lesion of the optic chiasm, or of the optic nerves anterior to the chiasm. Surgery may at times give dramatic improvement, or again the results may be equivocal or a failure.

A review of the author's experience discloses that over a period of ten years there have been 43 intracranial explorations in which no neoplasm or abscess was found and the diagnosis of serous meningitis or its equivalent was made (Table II). Some of these patients have not been followed and may later have shown definite evidence of cerebral tumor. However, a considerable group have been followed long enough to be reasonably certain that a space-occupying lesion does not exist. The majority of the patients have

### TABLE I. Classification of serous meningitis (intracranial)

#### A. LOCATION

1. GENERALIZED—serous arachnoiditis

2. LOCALIZED
   a) Cerebral hemisphere—often associated with a localized encephalitis—meningoencephalitis
   b) Optic chiasm—opticochiasmal arachnoiditis
   c) Posterior cistern—cisternal arachnoiditis

#### B. ETIOLOGY

1. OTITIC—otitis media—mastoiditis—sinusitis
2. TRAUMATIC—(subarachnoid hemorrhage?)
3. TOXIC—focus of infection at some distant point
4. UNKNOWN

#### C. TYPE

1. ACUTE OR SUBACUTE—inflammatory
2. CHRONIC—fibrous or proliferative (hyperplastic)
shown symptomatic improvement after operation; at times this has been dramatic. On the other hand, in many cases it could be reasonably argued that they might have done equally well without operation. In some (Case 5) the patient was temporarily improved, with later return and even exacerbation of symptoms.

Most of the deaths occurred in the cases of posterior fossa involvement, as there were five fatalities in the nine cases of this group that were operated on.

In this series, as well as in most reported, there is an unfortunate lack of pathologic studies. This is partly explained by the difficulty of getting adequate biopsies at operation. The report of Davis and Haven is in marked contrast to most of the literature, as these authors studied the arachnoid membrane in ten cases of this type. They divided their cases into three groups: the first, inflammatory (1 case); the second, fibrous (7 cases); and the third, hyperplastic (2 cases). In the last group cellular infiltration of the arachnoid was so marked that the authors raised the question of a possible neoplasm. Both of these patients were still living and well at the time of the report.

Table I represents an attempt at classification of the various intracranial types of serous meningitis. It is not original but rather is a combination of various classifications advanced by several authors, especially Claude, Horrax, Howell, Warrington, and Davis and Haven. It does attempt to separate the various classifications which have been made into comparable headings.

The great need is for more adequate correlation of microscopic findings with clinical types of serous meningitis. The careful study of individual cases may seem unproductive and disappointing but if persisted in should eventually shed further light on this obscure syndrome.

### REFERENCES


### TABLE II. Classification of author’s cases

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<th>Generalized serous arachnoiditis</th>
<th>Cases</th>
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<td>Cerebral hemisphere (meningoencephalitis)</td>
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<td><strong>TOTAL</strong></td>
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